"CLINICAL AND ULTRASONOLOGICAL PARAMETERS AS PRE-OPERATIVE INDICATORS OF DIFFICULT LAPAROSCOPIC CHOLECYSTECTOMY"

DISSERTATION SUBMITTED IN PARTIAL FULFILLMENT OF REGULATION FOR THE AWARD OF

M.S. DEGREE IN GENERAL SURGERY (BRANCH-1)

THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY

CHENNAI

APRIL 2014

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Assignment title	Medical
Author	22111225 . M.s. General Surgery THIRUVALAR PRABU ANAND V . VIKRAMADHITHAN
E-mail	vtpanand@gmail.com
Submission time	14-Dec-2013 07:20AM
Total words	17091

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ORIGINALITY REPORT

ACKNOWLEGEMENT

It is my privilege to express my sincere thanks to **Dr. Vimala, M.D**, Dean Coimbatore Medical College for permitting me to utilize the clinical materials of this hospital. It gives me immense pleasure to express my deep sense of gratitude and sincere thanks to my guide **Prof. Dr. S. Saradha M.S., Professor of Surgery** for her guidance, suggestions, advice and constant encouragement during the course of my study.

My sincere thanks to **Prof. Dr. V. Elango M.S., FIAS.,** Head of Department of General Surgery for his valuable guidance.

My heartfelt gratitude to **Prof. Dr. P.Swaminathan M.S.,, Prof. Dr. D.N. Ranganathan M.S., Prof. Dr. S. Natarajan M.S., Prof. Dr. G. Ravindran M.S., Prof. Dr. Balasubramanium M.S., Prof. Dr. P.V. Vasantha Kumar, M.S.,(Retd)**

I am thankful to Assistant Professors **Dr. N. Tamil Selan M.S., Dr. T.Srinivasan M.S., Dr. Murugesan M.S., Dr. Angeline Vincent M.S.,** and **Dr. R. Jaya Kuamr M.S.,** for their support and guidance. I thank all the assistant professors for their valuable inputs.

I thank my colleagues, CRRIs and staff nurses who have been a source of constant help. I am indebted to my patients who have submitted themselves to this study. I am grateful to my family who are a constant source of inspiration and support.

LIST OF ABBREVIATIONS

BMI -Body Mass Index

CBD -Common Bile Duct

ERCP - Endoscopic Retrograde Cholangiopancreatography

ESWL - Extracorporeal shock Wave Lithotripsy

ECG -Electrocardiogram

FBS -Fasting Blood Sugar

GB -Gall Bladder

LFT -Liver Function Test

LC -Laparoscopic Cholecystectomy

N -Normal

OC -Open cholecystectomy

IOC -Intraoperative cholangiography

OCG -Oral Cholecystography

PTC -Percutaneous Transhepatic Cholangiography

P value -Predictive value

PT-INR -Prothrombin time international normalized ratio

SGOT -Serum Glutamic Oxaloacetic Transaminase

SGPT -Serum Glutamic Pyruvate Transaminase

USG -Ultrasonography

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"CLINICAL AND ULTRASONOLOGICAL PARAMETERS AS PRE-OPERATIVE INDICATORS OF DIFFICULT LAPAROSCOPIC CHOLECYSTECTOMY"

ABSTRACT

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Pre-operative prediction of a difficult laparoscopic cholecystectomy (LC) can help the patient as well as the surgeon prepare better for the intra-operative risk and the risk of conversion to open cholecystectomy. **Methods:** In 50 eligible patients who went Laparoscopic cholecystectomy from November 2012 to November 2013, patient characteristics, Ultrasonological parameters and operative details were prospectively analysed to determine the pre-operative indicators of difficult LC. **Results:** Of the 50 patients, the parameters that significantly predicted difficult surgery were Thickened gall bladder wall (>3mm), contracted gall bladder, stone size >1 cm ($p<0.001$) and to some extent BMI ($p=0.05$) and Male gender. **Conclusion:** These results demonstrate that difficult laparoscopic cholecystectomy can be predicted based on parameters available preoperatively. Improvements in the ability to determine the risk for difficult surgery and conversion have important implications for surgical care.

KEY WORDS: LAPAROSCOPIC CHOLECYSTECTOMY, PREDICTIVE FACTORS, DIFFICULT LC, CONVERSION TO OC, PRE-OPERATIVE INDICATORS

INTRODUCTION

Cholelithiasis is the most common biliary pathology. Gallstones are present in 10 to 15% of the general population and asymptomatic in the majority (>80%).[1]The prevalence of gallstone varies widely in different parts of the world. In India it is estimated to be around 4%. An epidemiological study restricted to rail road workers showed that north Indians have 7 times higher occurrence of gallstones as compared to south Indians.[1] It is estimated that at least 20 million people in the United States have gallstones and that approximately 1 million new cases of cholelithiasis develop each year. Changing incidence in India is mainly attributed to westernization and availability of investigation that is ultrasound in both rural and urban areas and due to change in socioeconomic structure. Approximately 1-2% of asymptomatic patients will develop symptoms requiring cholecystectomy per year, making cholecystectomy one of the most common operations performed by general surgeons.[2]

Cholelithiasis is rare in the first two decades. Incidence gradually increases after 21 years and reaches its peak in 5th and 6th decade.[3] Women are more affected than men in the ratio of 4:1.[4]

In 1992, The National Institute of Health (NIH) consensus development conference stated that laparoscopic cholecystectomy “provides a safe and effective treatment for most patients with symptomatic gallstones.”[1] Since the

introduction of laparoscopic cholecystectomy, the number of cholecystectomy performed in the United States has increased from 5 lakh per year to 7 lakh per year.[2]

The advantages of laparoscopic cholecystectomy over open cholecystectomy are earlier return to bowel functions, less postoperative pain, informed cosmesis, shorter length of hospital stay, earlier return to full activity, and decreased overall cost.[47,48,49] Laparoscopic cholecystectomy is associated with better preservation of immune function and a reduction of the inflammatory response compared with open surgery. The rate of postoperative infections seems to be lower.[4]

Laparoscopic cholecystectomy has become the gold standard in the treatment of cholelithiasis and is replacing open cholecystectomy. The rate of conversion from laparoscopic cholecystectomy to open cholecystectomy is 5 to 10%.[5] Hence it is necessary to study the predictive factors for difficult laparoscopic cholecystectomy. Therefore this study was undertaken.

In this prospective study done in Dept. of General Surgery, Coimbatore Medical College, 50 patients suffering from symptomatic cholelithiasis are evaluated using specific clinical and ultrasonographic parameters prior to Laparoscopic Cholecystectomy to assess whether the difficulty of the procedure can be predicted, over a period of 1 year. It would be useful to accurately identify a patient's risk for difficult cholecystectomy based on pre-operative details and can result in accurate preoperative patient counselling, better

scheduling of surgery and appropriate assignment of surgical assistance, can increase the patient safety by reducing the time to conversion, and improving the mental preparedness of surgeons and patients also.

AIMS AND OBJECTIVES

STUDY GOALS:

The aim of this study is to evaluate the role of clinical and pre-operative abdominal ultrasonogram parameters as predictors of intra-operative difficulties and complications faced during Laparoscopic cholecystectomy.

OBJECTIVES:

- ✓ To study whether the specific clinical characteristics in patient with symptomatic cholelithiasis undergoing laparoscopic cholecystectomy like Age, Gender, BMI, Previous surgeries have any relation on the difficulties faced during LC.

- ✓ To study whether the pre-operative clinical and ultrasonography findings help predict the difficulty of LC in terms of duration of surgery, bleeding during LC, Gall Bladder bed dissection, difficulty in extraction, and conversion to Open Cholecystectomy.

REVIEW OF LITERATURE

HISTORICAL ASPECTS

The Roman celsus mentioned about the anatomic location of liver, De Medicina, as “The liver, which starts from the actual partition under the precordia on the right side, is concave within (that is on the inferior surface) and convex without; its projecting part rests lightly on the stomach and it is divided into four lobes. Outside its lower part, the gallbladder adhers to it”.[7]

Rhazes and Abicenna described the gallbladder, and stated in his treatise, Gallbladder- Causes and Treatment, “Vesalius found that he had a hemoperitoneum coming from an abscess which had eroded the portal vein. The gallbladder was yellow and contained 18 calculi. Very light, of a triangular shape wit even edges and surfaces everywhere, green by colour somewhat blackish. The spleen was very large.”

In 1562, stones in the gallbladder and common bile duct were described be Falloppio.

Fernel, in 1588, proposed that stasis was the predisposing factor for gallstone formation. He also noticed that in jaundice, the stool becomes pale and urine becomes darker, and the stones can be passed out via naturalis.

In 1769, Morgagni published an analysis of disease of the liver and biliary tract, in which he wrote about the incidence of stones in female and male patients.

Papilla of the duodenum was first described by Vater (1684-1751) in 1720; he described it as “those double ducts (bile and pancreatic ducts)…come together in single combination.”

In 1927 Wieland earned the Nobel Prize for describing the structure of the bile acids and in 1928, Windaus was awarded with Nobel prize for demonstrating the relationship between steroids and bile acids.

Pettit coined the term “biliary colic”

Zambecarri in 1630 and Techoff in 1667 had performed cholecystostomy and cholecystectomy in canine and showed that gall bladder is not essential for life.

Bobbs began the biliary surgery in 1867 in Indiana. He performed surgery on a women suspected to have ovarian cyst and was amazed to find an enlarged gallbladder filled with stones. He opened it, extracted the calculi and closed it with sutures.

Kocher, Simms and Trait operated on gallbladder affixing it anterior abdominal wall to allow the extraction of stones and pus and was left open to the exterior so that the peritonitis could be avoided.

1878: Kocher performed cholecystectomy in two stages. First, he packed the wound with gauze to the bottom of the gallbladder, and later after eight days, he removed the residual gallbladder stones.

1885: for the first time Trait performed cholecystectomy for the gallbladder lithiasis in one stage.

1882: Langenbuch performed the first elective cholecystectomy and in the same year Von Winiwarter developed cholecystoenterostomy.

1895: Kocher wrote e on internal choledochoduodenostomy which was done to retrieve supra-ampullary choledochal stones.

1897: First systemic use of biliary intubation was done by Kehr when he introduced a rubber tube through cystic duct into the common bile buct..

1898: Thornton was the first surgeon to remove commom bile duct calculi.

Sprengel in 1891 and Riedel in 1892 were the first to do supraduodenal choledochoduodenostomy. Sprengel performed a side-to-side choledocho-duodenostomy, which later became famous in Europe and United States and by many surgeons like Oddi (1888).

1898: Mc Burney published about papillotomy and duodenostomy in patients with impacted calculi in periampullary region.

1898: Buxbaum observed biliary calculi on plain x-rays.

1912: T-tube was developed by Kehr.

1923: Choledochoscopy was developed by Bakes.

1924: Graham developed oral cholecystography.

1932: Mirizzi developed postoperative cholangiography. And in **1937** he developed intra-operative cholangiography.

1989: Dubois (Dubois et al) published the first series of laparoscopic cholecystectomy.

HISTORY OF LAPAROSCOPY AND LAPAROSCOPIC CHOLECYSTECTOMY:

The word laparoscopy is a Greek word, “laparo” meaning the flank and “skopein” meaning to examine. Laparoscopy was first performed by George Killing of Dresden, Germany in 1901 on dog, using room air filtered through sterile cotton for pneumoperitoneum and a wide cystoscope to view the abdominal cavity. In 1924, Richard Zollikofer of Switzerland first suggested the use of carbon dioxide (co2) for creating pneumoperitoneum. Janos Veress of Hungary in 1938 introduced the primary mode of insufflation by Veress needle.

A dual trochar laparoscopic technique for liver biopsy was described by German hepatologist Kalk in 1938.

Feowers, a German general surgeon, was the first to report laparoscopic lysis of abdominal adhesions for the diagnosis of bowel obstructions.

Laparoscopic tubal ligation was done by Patrick Steptoe in 1969 and made it popular.

Kurt Semn incorporated the new aspects of fibre optic and used automatic gas insufflators which allowed precise controlled intra abdominal pressure.

Laparoscopic cholecystectomy was described for acute cholecystitis by Lukichev and colleagues in 1983. In 1985, the first laparoscopic assisted cholecystectomy was done by Muhe of Boblinger , Germeny. The first video laparoscopic cholecystectomy was performed by Phillipe Mouret, a French surgeon in Lyon.

ANATOMY:

The extra hepatic biliary tree consists of the left and right hepatic ducts, common hepatic duct, gall bladder, cystic duct and the common bile duct.

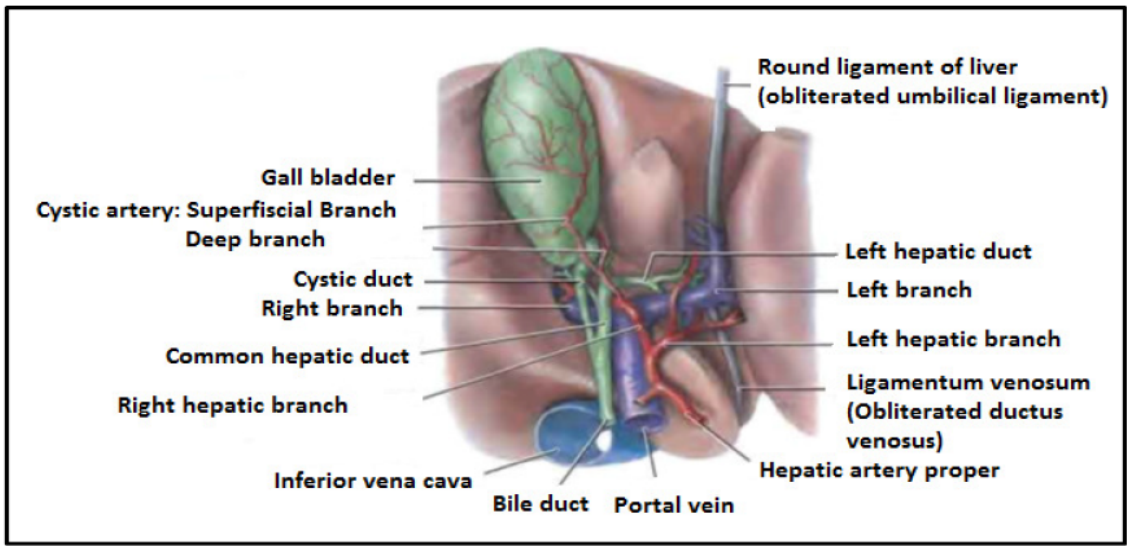


FIGURE 1: Anatomy of gallbladder, inferior view.

GALLBLADDER:

The gallbladder is a blind-ending diverticulum which is flask-shaped, draining into the common bile duct via the cystic duct. In adult human its size is usually between 7-10cm, with a capacity of 50 ml.[3] it is grey-blue in colour and usually lies in a shallow fossa of liver parenchyma covered by the peritoneum continuing from the liver surface, and is attached to the inferior surface of the right lobe of the liver by connective tissue. This attachment of the GB varies widely. At one extreme it shows "INTRAPARENCHYMAL PATTERN" where the gallbladder is almost embedded within the liver surface, with no peritoneal covering. And at the other end it has "MESENTRIC PATTERN", where it hangs out from a short mesentery made up of two layers of peritoneum separated by small vessels and connective tissue.[8] The parts of a gallbladder are: fundus, body and neck. The gallbladder lies on a fibrous or cystic plate known as the cystic plate, which forms the part of the peri-hilar system of fibrous tissue. The cystic plate attaches directly to the anterior surface of the right portal pedicle. The hepatic parenchyma lies deep to this cystic plate, through which these small bile ducts of Luschka (consisting of accessory ducts <1mm in diameter) may penetrate to enter the gallbladder. In approximately 10% of people, there is a large peripheral duct immediately deep to this plate, which if dissected may cause bile leakage.[9] The origin of middle hepatic vein lies at variable depths beneath the cystic plate and may enter inadvertently.

There is thin layer of areolar tissue present between the muscularis of gallbladder and the cystic plate, which gets thickened from going top to bottom of gallbladder. During dissection of gallbladder from the liver, when the areolar tissue is left on the cystic plate, the posterior surface of the cystic artery and bile duct will be reached. If the dissection is done deep into the cystic plate, the surface of the right portal pedicle may be breeched and result in injury to the right portal pedicle structures and the right hepatic duct.[10]

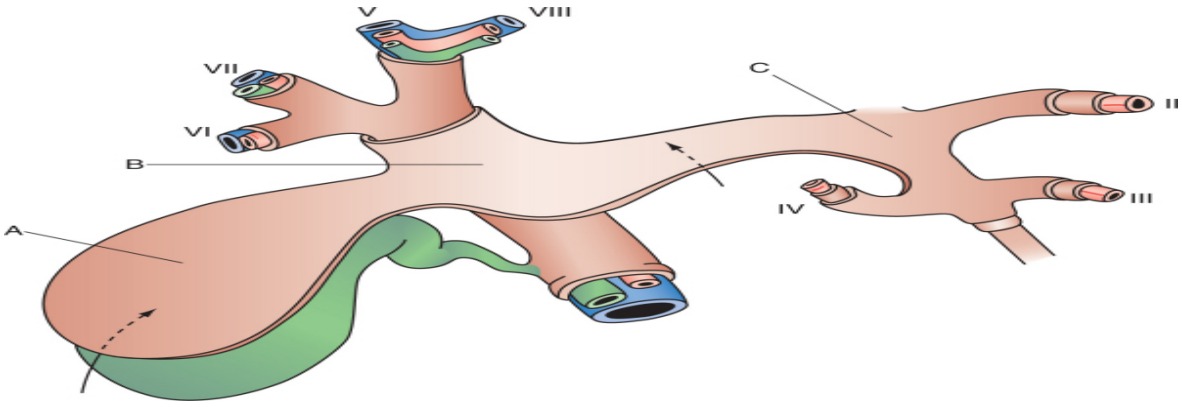


FIGURE 2: Plate system anatomy. Cystic plate (A) the hilar plate (B) the umbilical plate (c)

NECK:

Neck of the gall bladder is at the medial end of porta hepatis, and anterior to the second part of duodenum, and is almost always has a short peritoneal cover attached to liver called as MESENTRY, which usually contains the cystic artery. At the medial end, mucosa is ridged obliquely which forms a spiral groove continuous with the spiral valves of cystic duct. At its lateral end the

neck widens to form the body and this part of gall bladder is known as "HARTMANN'S POUCH".[11]

BODY AND FUNDUS:

The body of the gallbladder usually lies in contact with the liver surface. It lies anterior to the 2nd part or the duodenum and the right end of the transverse colon. The fundus lies at the lateral end of the body and projects out the inferior border of the liver to a variable length. It lies in contact with the anterior abdominal wall behind the 9th costal cartilage, where the lateral edge of the rectus abdominus crosses the costal margin. At this location the enlargement of the gallbladder is best sought on clinical examination. Sometimes the fundus of gallbladder is folded back upon the body of gallbladder known as "PHRYGIAN CAP".[11]

EXTRAHEPATIC BILIARY TREE

CYSTIC DUCT:

The cystic duct is about 3 to 4 cm in length, passes posteriorly to the left from the neck of the gallbladder, and joins the common hepatic duct to form the common bile duct. It almost runs parallel to it and is adherent to the common hepatic duct for a short distance before joining it. The junction usually occurs near the porta hepatis but may also be present lower down in the free border of the lesser omentum.

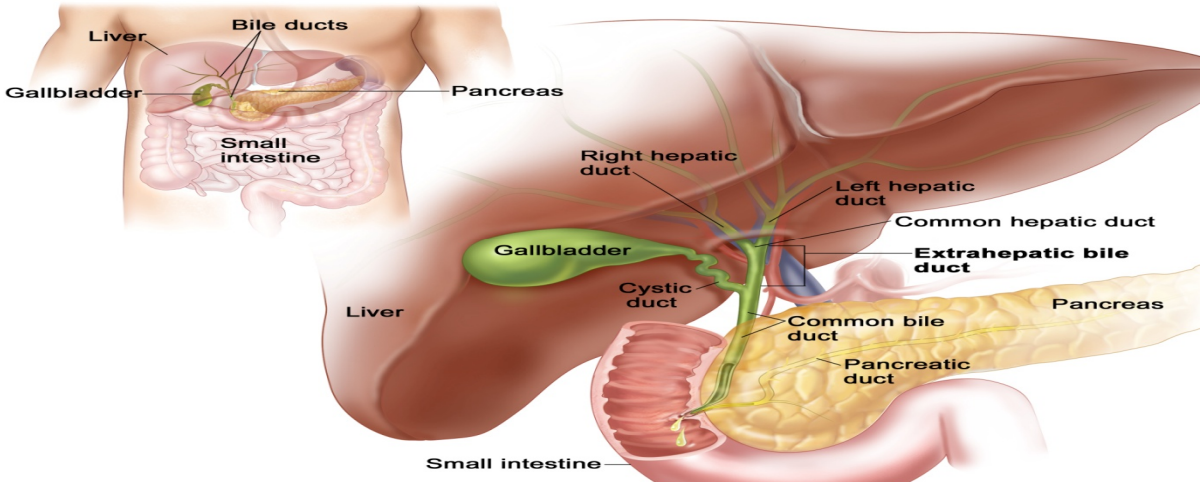


FIG. 3**:** Showing the anatomy of the gallbladder, biliary radicals, pancreatic duct and the hepato-pancreatic ampulla.

ANATOMICAL VARIATIONS OF CYSTIC DUCT: [60]

1) The cystic duct may be elongated, lying posterior or anterior to common hepatic duct draining into the left border of right hepatic duct.
2) Cystic duct may be closely adherent to common bile duct where the right edge of the lesser omentum lodges the cystic duct, going all the way to the duodenum where it forms the junction.
3) The cystic duct may be double or absent. If it is absent, then the gallbladder drains directly into common bile duct.
4) One or more accessory hepatic ducts occasionally emerge from the V segment of the liver and joins either the right hepatic duct, the common hepatic duct, the common bile duct, the cystic duct or the gallbladder.

The mucosa of the cystic duct bears 5-12 crescentic folds which is continuous with those present in the neck of gallbladder.[50] If the duct is cut

in longitudinal section these crescentic folds project obliquely in regular succession appearing to form a spiral valve. When the duct is distended the spaces between the folds dilate and externally it appears twisted like the neck of the gallbladder.[59]

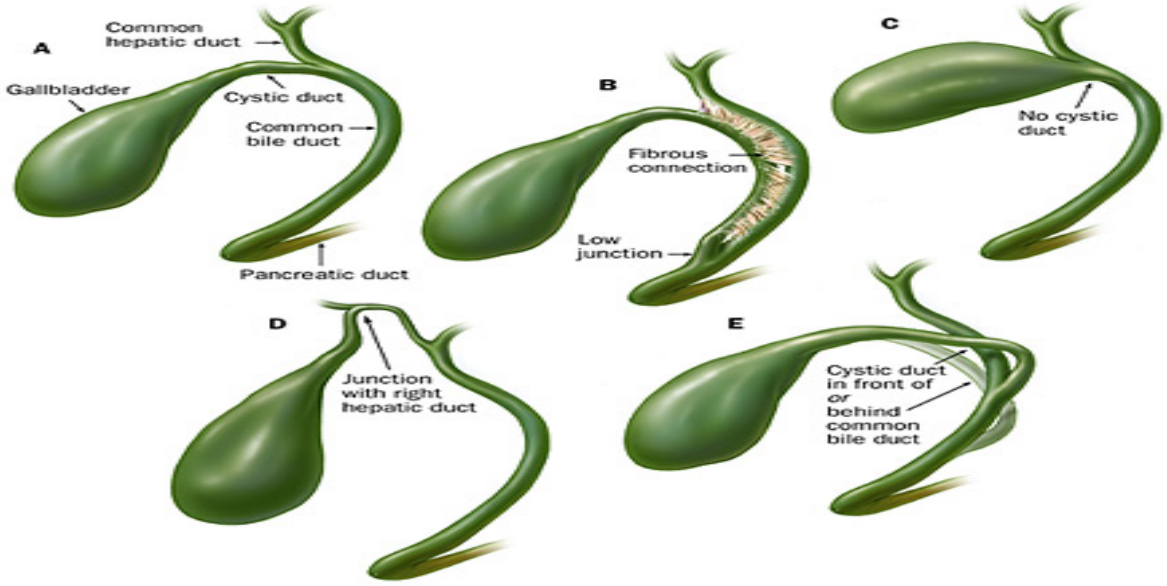


FIG.4 Showing Variations in Cystic Duct

HEPATIC DUCTS:

The main right and left hepatic ducts emerging from liver unites to form common hepatic duct near the right end of porta hepatis. It descends down for about 3cm before it joins the cystic duct at an acute angle to form common bile duct. The common hepatic duct lies in the free edge of lesser omentum where it is at right of hepatic artery and anterior to the portal vein.

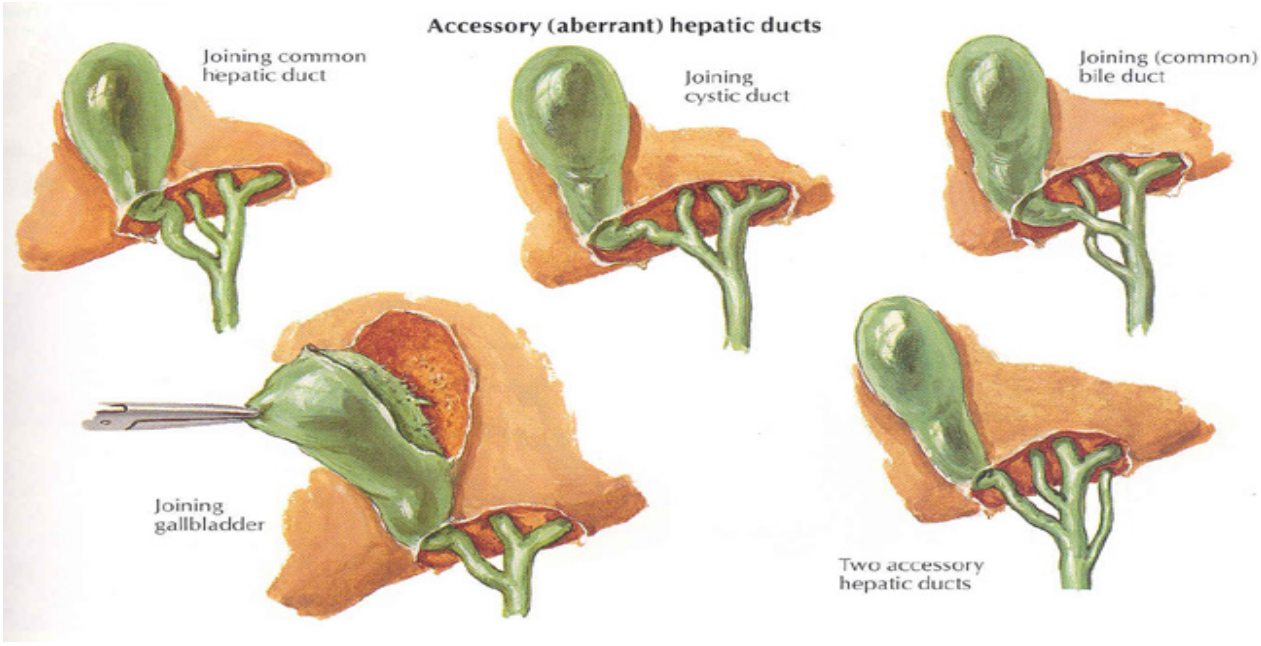


Fig.5 Showing Variations in Hepatic Duct

COMMON BILE DUCT:

Common bile duct is formed by the junction of cystic and common hepatic ducts near the porta hepatis. In adults its length is between 6-8cm and diameter of about 6mm.[12] It lies anterior & to the right of the portal vein and to the right of the hepatic artery. It passes behind the first part of the duodenum with gastroduodenal artery on its left and then runs in a groove on the supero-lateral part of the posterior surface of the pancreas. The duct may lie close to the medial wall of the 2nd part of the duodenum or as much as 2cm from it.

HEPTOPANCREATIC AMPULLA (OF VATER):

It is formed by the union of CBD and pancreatic duct before entering the second part of the duodenum. Circular muscles surrounds the lower part of CBD to form “bile duct sphincter”, it also surrounds the terminal part of the

main pancreatic duct to form “pancreatic duct sphincter”, and the hepatopancreatic ampulla to form “sphincter of oddi”.[59]

CALOT’S TRIANGLE (CHOLECYSTOHEPATIC TRIANGLE)

The triangular space formed by the borders of cystic duct, common hepatic duct and the inferior surface of the segment V of the liver is referred to as Calot’s triangle. A double layer of triangle encloses the triangle which forms the short mesentery of cystic duct.[59]

It is better described as a pyramidal space with one apex at the junction of cystic duct and fundus of the gallbladder, one at the porta hepatis and two closer apices at the attachment of gallbladder to the liver bed. The inferior surface of the liver thus forms the base of the triangle.[60]

CONTENTS OF THE CALOT’S TRIANGLE

1) Cystic artery as it approaches the GB.
2) Cystic lymph node.
3) Lymphatics from the GB.
4) 1 or 2 small cystic veins.
5) Autonomic nerves running to the GB.
6) Some adipose tissue.
7) Accessory ducts which may drain into GB from liver.

VASCULAR SUPPLY AND LYMPHATIC DRAINAGE: [60]

CYSTIC ARTERY:

The cystic artery originates from the right hepatic artery. It reaches the superior aspects of the neck of bladder by passing behind the common hepatic duct and over the cystic duct. It divides into superficial and deep branches, where the superficial branches ramify on the inferior aspect of the gallbladder, and the deep branches on the superior aspect. These arteries anastomose over the surface of the body and fundus. The cystic artery is the end artery and its occlusion causes gangrene of the gallbladder.

ANATOMICAL VARIATIONS: [60]

1) It may arise from the common hepatic artery, sometimes from the left hepatic artery or rarely from the gastroduodenal or superior mesenteric arteries, where it may cross anterior to the CBD or CHD to reach gall bladder.
2) An accessory artery may arise from the common hepatic artery or one of its branches.
3) The cystic artery often bifurcates at its origin to give rise to two arteries supplying gallbladder.
4) Multiple fine arterial branches may arise from segment IV or V and contribute in supplying the body of gallbladder. The cystic artery gives

rise to multiple fine branches which supply the common and lobar hepatic ducts and the upper part of CBD.

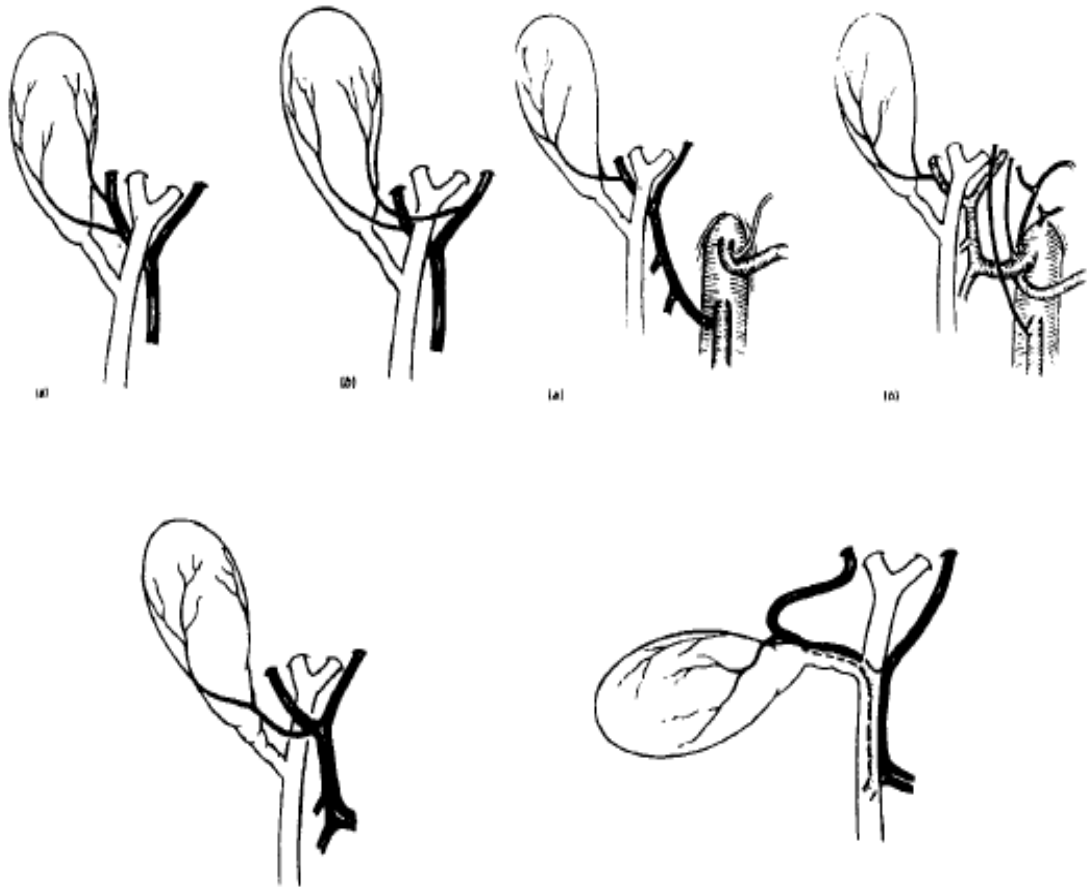


Fig 4: Vascular anomalies

(a). Accessory cystic arteries – arising from right hepatic artery.

(b). Accessory cystic artery – arising from left hepatic artery.

(c). Hepatic artery arising from superior mesenteric artery.

(d). Accessory hepatic arteries arising from the coelic trunk and superior mesenteric arteries

(e). Anterior transposition of right hepatic artery and cystic artery

(f). Recurrent (caterpillar hump) right hepatic artery.

Fig. 6 Showing Vascular anomalies of Gall Bladder

DUCTAL ARTERIES:

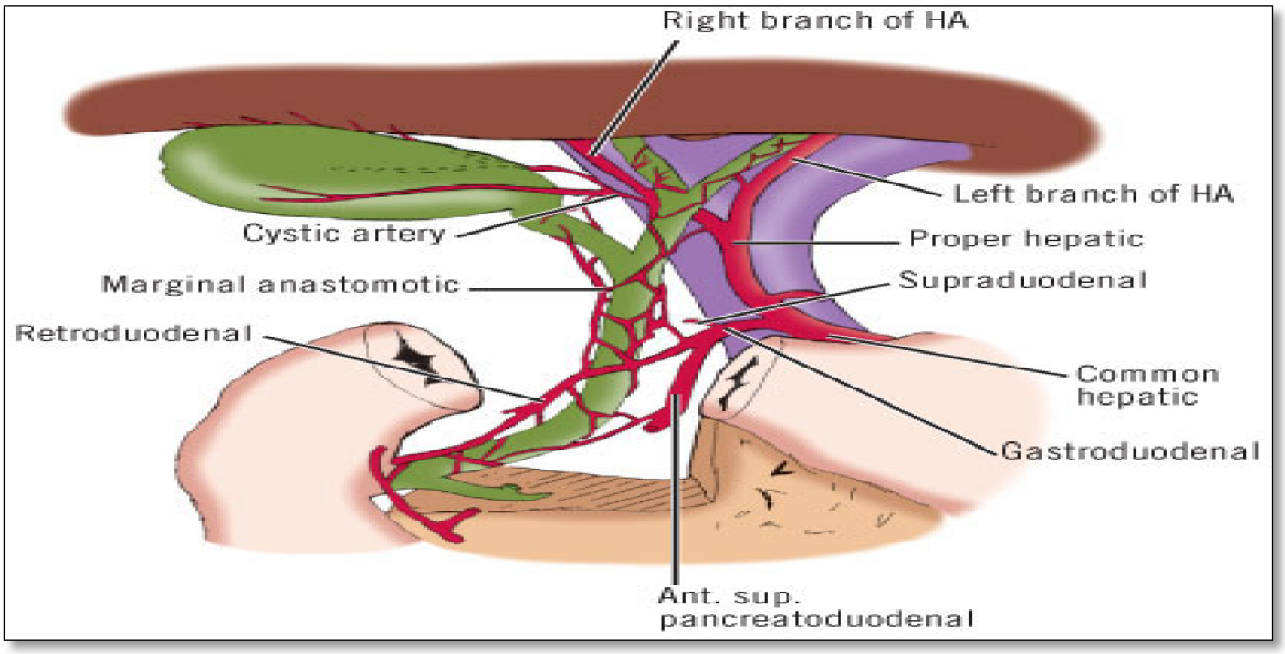


Fig. 7 Blood supply of ductal system of biliary tract

The hepatic ducts and common bile duct are supplied by the fine network of vessels, which lie in close proximity to the ducts themselves. During surgical exposure of the bile ducts over a long length, causing any disruption of the network causes chronic ischemia and stenosis.

Anterior to CBD, 2-4 ascending vessels arise from the retro duodenal part of the gastro duodenal artery. 3-4 descending branches of the right hepatic and cystic arteries arise as this vessel pass close to lower CHD.[64]

These descending and ascending arteries form long narrow anastomotic channels along the length of the duct called “medial” and “lateral trunks”.

Posteriorly, a retroperitoneal artery often arises from the coeliac axis, superior mesenteric artery or one of its major branches close to its origin from the aorta. It runs upward on the posterior surface of the portal vein, where it contributes in forming the arterial network supplying the supraduodenal part of the bile duct system.

CYSTIC VEINS:

The veins lying in the areoler tissue between the gallbladder and the liver are those which are arising from the superior surface of the body and neck, and it enter the liver parenchyma to drain into the segmental portal veins. The remaining forms 1-2 cystic veins which enter the liver directly or after joining the veins draining the hepatic ducts and upper bile ducts. Sometimes rarely a single or double cystic vein drain into the right portal branch.

LYMPHATICS:

There are numerous lymphatic vessels running from the submucosal and subserosal plexus on all aspects of the gallbladder and cystic duct. The one present on the hepatic aspect of the gallbladder connect with the intrahepatic lymphatics. The remaining drains into the cystic node, lying above the cystic duct in the tissue of Calot’s triangle. This node along with the some of the lymphatic channels which bypass the cystic node, drain into a node lying in the anterior border of the free edge of the lesser omentum.

INNERVATION:

Branches from the hepatic plexuses innervate the gallbladder and the extrahepatic biliary tree. The pyloric branch of vagus innervates the retroduodenal part of CBD along with the innervations to the smooth muscle of the hepatopancreatic ampulla.

REFERRED PAIN:

Pain from the stretching of the CBD or gallbladder is referred to the central epigastrium. Involvement of the overlying somatic peritoneum produces pain which is more localised to right quadrant.[14]

EMBRYOLOGY:

At the middle of third week an endodermal epithelium outgrowth at the distal end of foregut appears which forms "liver primordium"[15, 16]. This outgrowth known as hepatic diverticulum or the hepatic bud consists of rapidly dividing cells that penetrate the septum transversum. As the hepatic cells continue to penetrate, the connection between the hepatic diverticulum and the foregut narrows forming the bile ducts.

On day 26, cystic diverticulum develops as a distinct endodermal thickening which appears on the ventral side of the duodenum just caudal to the base of the hepatic diverticulum.[17] This cystic diverticulum forms the

gallbladder and the cystic duct. CBD is formed by the proliferation of the cells at the junction of hepatic and cystic duct.

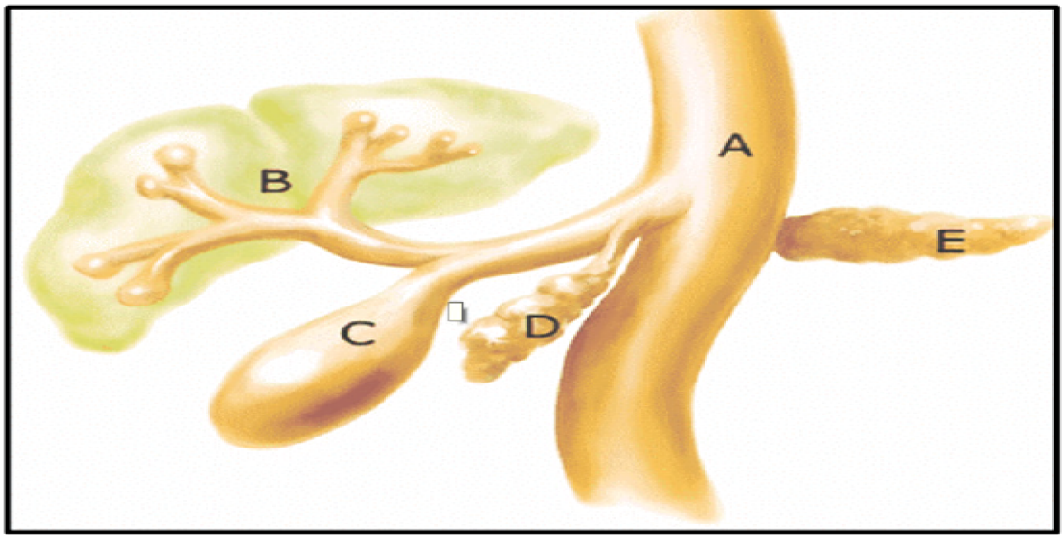


FIGURE 8: illustrating the foregut (A), the cranial end of the hepatic diverticulum which represents Pars hepatica (B) and the Cystic diverticulum (C). The ventral (D) and dorsal (E) pancreas are also demonstrated.

At 10th week of development the liver starts producing red blood cells and white blood cells due to formation of large number of sinusoids and nests of proliferating cells, which lies between the hepatic cell and the wall of the vessels.[19, 20] At 12th week of life, liver starts producing bile, which is dark green in colour.

HISTOLOGY:

GALLBLADDER:

The mucosa is yellowish-brown giving a honeycomb appearance because of minute rugae. These projections of mucosa into gallbladder lumen resembles intestinal villi, but these gets flattened as the gallbladder fills with the bile.[21]

The epithelium is a single layered columnar epithelium with apical microvilli, and goblet cells are absent. At the basal end the spaces between epithelial cells are dilated. Many capillaries lie beneath the basement membrane.

Beneath the basement membrane, there is a thin fibro-muscular layer which is composed of fibrous tissue mixed with smooth muscles which are arranged loosely in longitudinal, circular and oblique bundles.

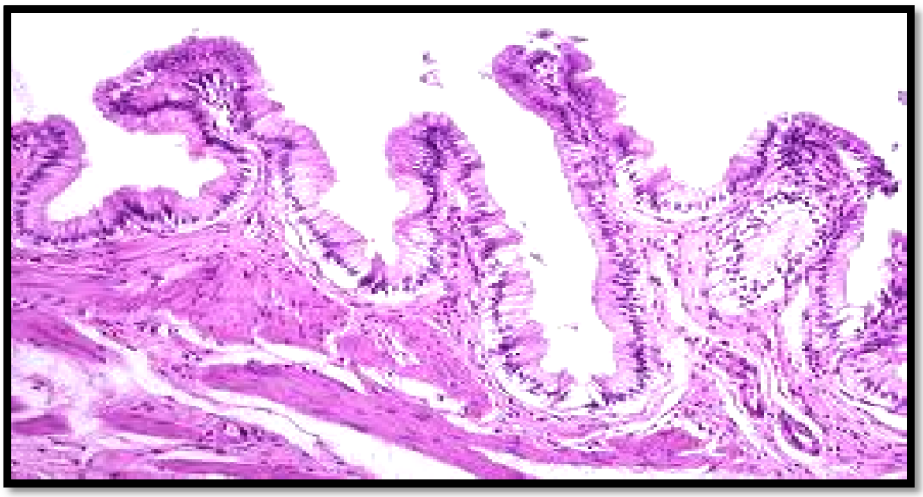


FIGURE 9: Histology of gall bladder wall.

BILE DUCTS:

The epithelial lining is columnar epithelium, containing mainly tubule-alveolar mucous glands. The larger biliary ducts have external fibrous and internal mucous layers. The fibrous connective tissue containing variable number of connective tissue and variable amount of longitudinal, oblique and circular smooth muscles.[23]

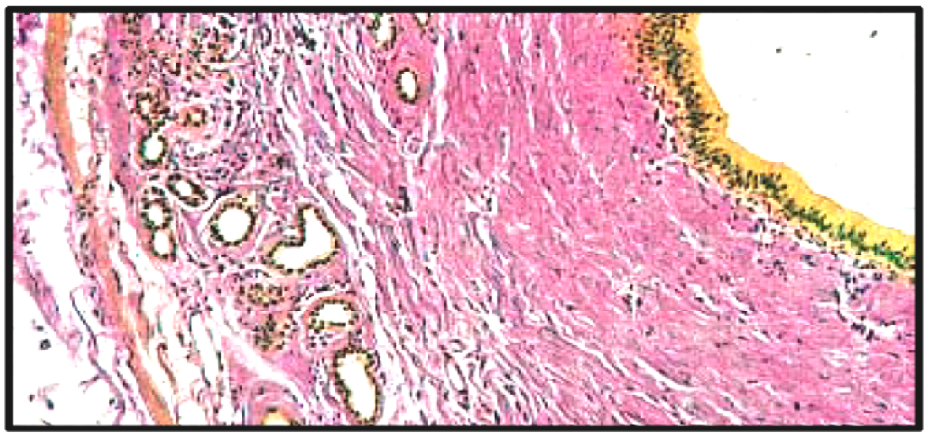


FIGURE 10: Histology of common bile duct.

PHYSIOLOGY:

Bile is made up of bile salts, bile pigments and other substance dissolved in alkaline medium. About 500ml is secreted daily. The golden yellow colour of the bile is formed due to glucoronides of the bile pigments, bilirubin and biliverdin.

Water	97.0%
Bile salts	0.7%
Bile pigments	0.2%
Cholesterol	0.06%
Inorganic salts	0.7%
Fatty acids	0.15%
Lecithin	0.1%
Fat	0.1%
Alkaline phosphatase	-------

Table.1 COMPOSITION OF HEPATIC BILE:

Bile acids are synthesized from cholesterol and contain cyclopentanoperhydrophenantherene nucleus.[24] The primary bile acids formed in liver are- cholic acid and chenodeoxycholic acid. These get converted into secondary bile acids in the colon by the bacteria which converts cholic acid to deoxycholic acid and chenodeoxycholic acid to lithocholic acid.

Bile salts are sodium and potassium salts of bile acids. These when secreted into bile are conjugated by glycine or taurine. They are amphipathic and forms micelles. Lipid when gets collected in the micelles, the cholesterol comes at the hydrophobic center and at the other hydrophilic end amphipathic phospholipids and monoglycerides lines up. The micelles play a very important role in keeping lipids in solution and transporting them to the brush border of the intestinal epithelial cells where they gets absorbed.

90-95% of bile salts are absorbed from small intestine. Some by nonionic diffusion and majority by Na+ - bile salt co-transport system powered by Na+ - K+ ATPase in terminal ileum

The remaining 5-10% enters into colon and are converted to the salts of deoxycholic acids lithocholic acids. Where lithocholate is insoluable and excreated in stools. While, deoxycholate is absorbed. These absorbed bile salts are transported back to the liver in portal vein through enterohepatic circulation and re-excreated in bile. Those lost in stools are replaced by synthesis in liver.

The bile salt is synthesised at the rate of 0.2-0.4g/day. The total bile salts pool of approximately 3.5g recycles repeatedly via EHC. Entire pool recycles twice per meal and 6-8 times per day.[25]

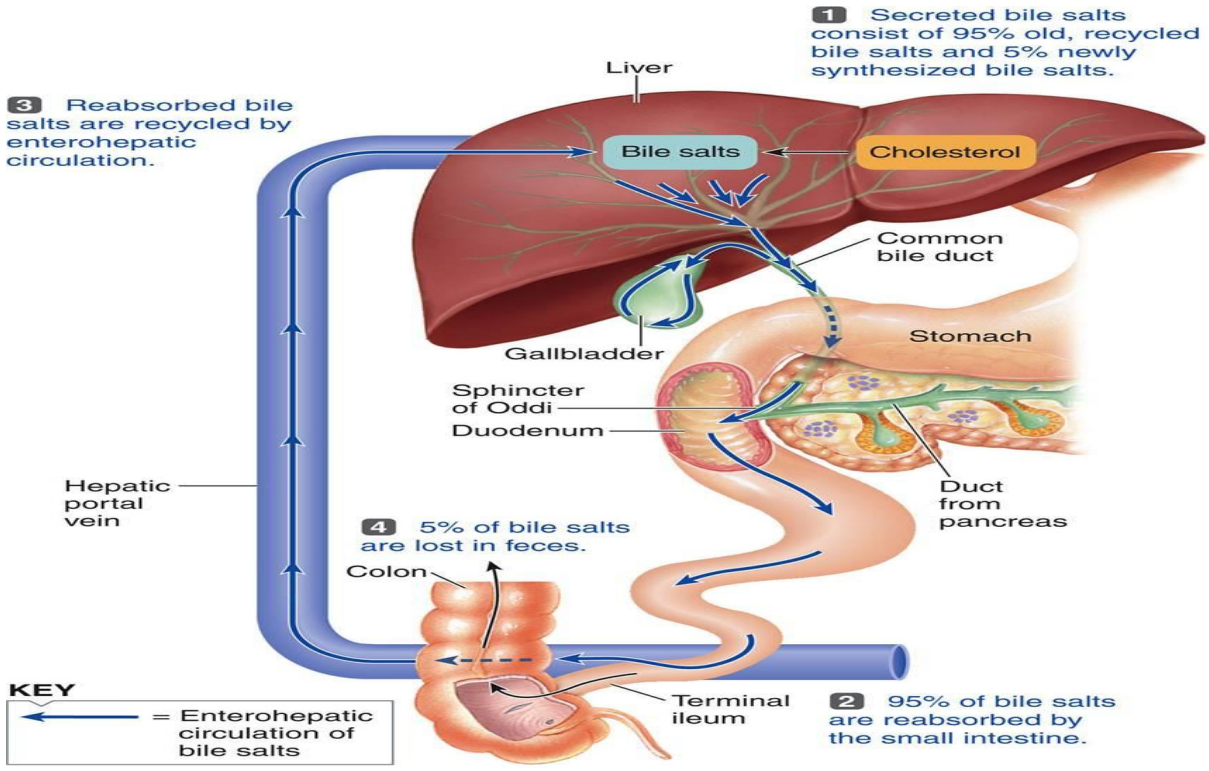


FIGURE 11: showing enterohepatic circulation of bile salts.

BILIRUBIN METABOLISM AND EXCRETION:

Bilirubin is formed by the breakdown of haemoglobin. In circulation bilirubin is bound to albumin which gets dissociated in liver where it gets bound to cytoplasmic protein in liver cells. For bilirubin to become soluble it has to get conjugated to glucuronic acid by UDP- glucuronyl transferase (located in endoplasmic reticulum), to form bilirubin diglucuronide, which gets transported actively against concentration gradient into bile canaliculi.[25, 26.] Some amount of

bilirubin glucuronide escapes into blood, where it gets bound to albumin and is excreted in urine. Similarly some amount of urobilinogen enters the general circulation through portal circulation and is excreted in urine.

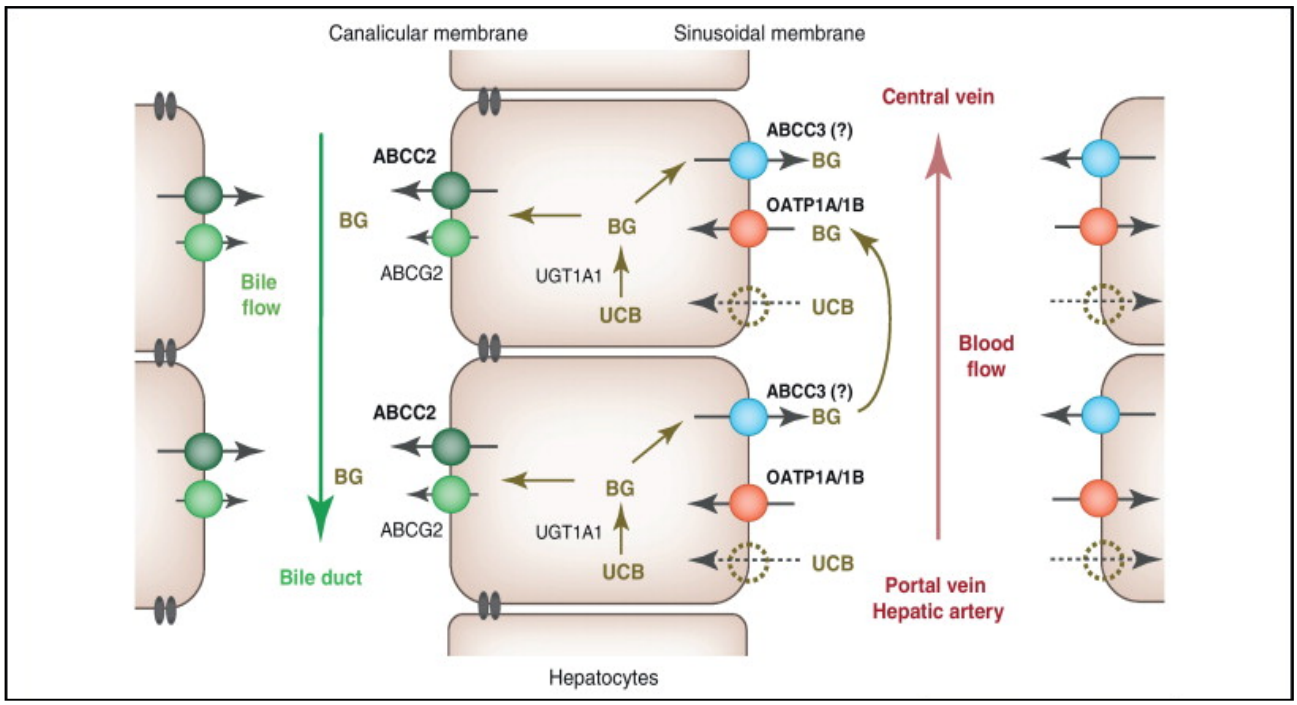


Fig.12 Showing Bilirubin Metabolism in Liver

REGULATION OF BILIARY SECRETION:

As soon as the food enters the mouth the tone of sphincter of Oddi gets decreased. The fatty acids and the amino acids (known as cholagogues) in the duodenum releases CCK, which cause gallbladder contraction. The production of bile is increase in by stimulation of vagus nerves and hormone “secretin”, which increases the water and HCO3- content of bile. Bile salts are the important cholerectics which increases the secretion of the bile.

PATHOGENESIS:

In western countries, about 80% are cholesterol stones, which contain 50% of crystalline cholesterol monohydrate. Remaining 20% are pigment stones, which are composed of bilirubin calcium.[3]

I. CHOLESTEROL STONES:

Cholesterol becomes soluble in bile by the detergent effect of water soluble bile salts and water insoluble lecithin. But when the super saturation of cholesterol occurs, it can't remain dispersed and nucleate into solid cholesterol monohydrate crystals.

DEFECTS INVOLVED IN THE FORMATION OF CHOLESTEROL GALLSTONES:

1) SUPERSATURATION OF BILE WITH CHOLESTEROL:

This is a primary defect, caused by the abnormal regulation of hepatic mechanisms for delivering cholesterol to bile. The excess of free cholesterol is toxic to gallbladder and starts penetrating the wall of gallbladder, exceeding its ability to detoxify by esterification. This causes the hypo mobility of gallbladder by the muscular stasis, which occurs due to both from dysmobility and decreased response of neuromuscular to CCK.

2) HYPOMOBILITY OF GALLBLADDER:

Hypomobility of gallbladder promotes enucleation.

3) ACCELERATION OF CHOLESTEROL NUCLEATION IN BILE:

It is due to shift in balance between antinucleating and pronucleating proteins and presence of micro precipitants of inorganic or organic calcium salts.

4) HYPERSECRETION OF MUCOUS:

Mucous hyper secretion in gallbladder traps the crystals, permitting their aggregation into stones.

Superimposed conditions that exacerbate defective gb emptying and cholesterol stone formation:

1) Prolonged fasting
2) Total parenteral nutrition
3) Pregnancy
4) Spinal cord injury
5) Rapid weight loss

II. PIGMENT STONES:

Pigment stones are complex mixtures of insoluble calcium salts of unconjugated bilirubin with inorganic calcium salts. Any infection of biliary tract with E.coli or Ascaris lumbricoids or by the liver flukes opisthorchis sinensis leads to the release of microbial β-glucuronidase, which hydrolyses bilirubin glucuronides to unconjugated bilirubin.[27]

On the other side, intravascular hemolysis leads to increased hepatic secretion of conjugated bilirubin. The aqueous solubility of free bilirubin may easily be exceeded in haemolytic conditions.

MORPHOLOGY:

I. CHOLESTEROL STONES:

They are exclusively found in gallbladder, composed of 50- 100% of cholesterol. In pure cholesterol stones, they have a fine granular, hard external surface which is pale yellow in colour, and round to ovoid in shape. It gives an appearance of glistening radiating crystalline palisade when a transection is done.

As the composition of calcium carbonate, phosphates and bilirubin increases, the stones become discoloured and appear gray white to black on transection. Mostly multiple stones of size ranging up to several centimeters in diameter are present. Surfaces of multiple stones may be rounded or faceted, to get tight apposition. Stones composing of cholesterol are radiolucent; while in 10-20% of stones containing sufficient calcium carbonate render them as radiopaque.

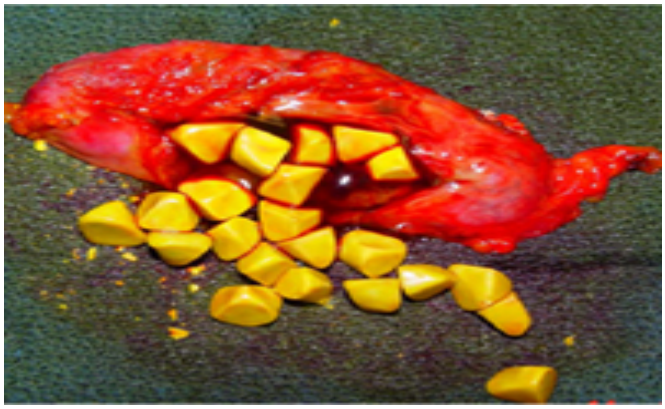


Fig. 14 Cholesterol Stones

II. PIGMENT STONES:

They are classified as brown and black stones.

a. Black stones:

It is found in sterile bile of gall bladder. It contains oxidized polymer of calcium salts of unconjugated bilirubin; some amount of calcium carbonate, calcium phosphate and mucin glycoproteins and a modicum of cholesterol monohydrate crystals. The size does not exceed more than 1.5cm in diameter, having a spiculated or moulded contours. They are present in large numbers and may crumble on touch. About 50-70% stones are radiopaque due to presence of calcium carbonates and phosphates.[45, 46]

Fig.15 Black pigment Stones

b. Brown stones:

They are found in infected intrahepatic and extrahepatic ducts. They contain pure calcium salts of unconjugated bilirubin, mucin glycoproteins, a substantial amount of cholesterol fraction and calcium salts of palmitate and stearate. They have soft and laminated appearance with soap like greasy consistency.

Fig.16 Brown Pigment stones

BILIARY SLUDGE:

It is a mixture of cholesterol crystals, calcium bilirubinate granules and mucin gel matrix.[29, 20, 31]. This suggests that sludge may serve as the nidus for gall stone pathogenesis. It is usually seen in prolonged fasting states or with the use of parenteral nutrition.

THE NATURAL HISTORY OF GALLSTONES

Gallstones are the most common digestive disease. It is estimated that 10-15% of the adult population (>20 million) in US had gallstones, during 1992. About 1 million patients are newly diagnosed annually, and around 600,000 patients underwent cholecystectomy in 1991.[28,29,50]

EPIDEMIOLOGY:

It is the most common gastrointestinal illness with a prevalence of 11- 36 % in autopsy reports. The strongest risk factors for its development are first degree relatives of the patients having gallstones and obesity (BMI > 30 kg/m^2).[29, 32]

RISK FACTORS FOR GALL STONES:

1) Obesity
2) First degree relatives
3) Rapid weight loss
4) Drugs : Ceftriaxone, postmenopausal estrogens

5) Total parenteral nutrition

6) Childbearing

7) Ethnicity : Native American, Scandinavian

8) Multiparity

9) Ileal disease- Resection or bypass.

10) Female sex

11) Increasing age

CLINICAL PRESENTATION

Gallstones are asymptomatic in most patients. Whereas in some patients, symptoms occurs as biliary colic, which is caused by a stone obstructing the cystic duct. In asymptomatic patients, only 1% to 2% develops serious symptoms or complication per year; hence only about 1% required cholecystectomy.[10, 12, 33]. When patients become symptomatic, they tend to have recurring symptoms, usually repeated episodes of biliary colic. 10 – 30% of patients develop non-specific gastrointestinal symptoms, 5 – 10% of patients develop classic biliary symptoms.[37]

BILIARY COLIC:

It is a misnomer, because the pain is not colicky in the epigastrium or right upper quadrant. Biliary colic is a constant pain that builds in intensity and

can radiate to the back, inter-scapular area or right shoulder. It results due to acute obstruction of gallbladder by calculi.

The pain is described as band- like tightness of the upper abdomen which is associated with nausea and vomiting. This is caused due to contraction of normal gallbladder against an obstructed lumen by an impacted gallstone in the neck of gallbladder, the cystic duct or CBD. The pain is most commonly triggered by fatty foods or may be any other types of food or even it can occur spontaneously. Only 50% of patient shows association of pain with meals, which often develops after more than an hour of eating.[36, 37]

INVESTIGATIONS:

1) **LIVER FUNCTION TEST:**

   Obstructive choledocholithiasis shows raised value of direct bilirubin and elevated alkaline phosphatase levels. Leucocytosis, predominantly neutrophils, are present in acute cholecystitis and cholangitis.

2) **PT – INR:**

   Prolonged prothombin time is present in liver dysfunction.

3) **ROUTINE BLOOD INVESTIGATIONS:**

   a) Complete hemogram

   b) Renal function tests

c) ECG

4) IMAGING STUDIES:

i. PLAIN RADIOGRAPHS:

Only 15% of gallstones containing enough calcium to make them radiopaque are visible on plain abdominal films.[38] It is usually done to rule out perforation with free intra-peritoneal air, bowel obstruction with dilated loops or right lower pneumonia.

ii. ULTRASONOGRAPHY:

It is a routine evaluation in patients with cholelithiasis having a sensitivity of >98%. USG not only identify the gallstones but also gives detail signs of cholecystitis such as:

- ✓ Thickening of gallbladder wall
- ✓ Pericholecystic fluid
- ✓ Impacted stones in neck of gallbladder.

Dilatation of extrahepatic (>10 mm) or intrahepatic (>4 mm) bile ducts suggests biliary obstruction.[38, 39]

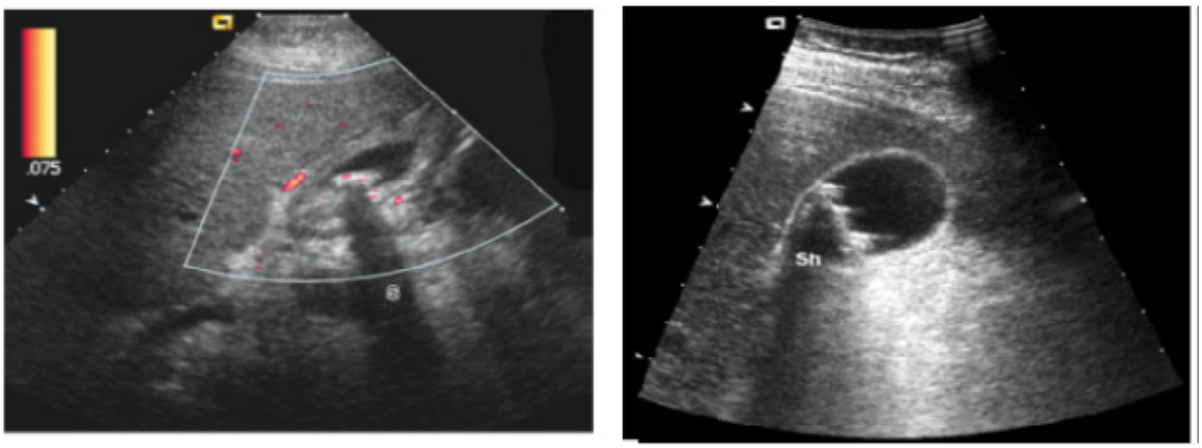


Fig 17 A, Echogenic foci in the gallbladder with acoustic shadowing (S) are characteristic of gallstones. In this patient, the gallbladder wall is thickened, but not hypervascular. Features suggest chronic cholecystitis. B, multiple stones are layered in the dependent portion of the gallbladder, but the wall is not thickened. Sh, shadow.

iii. ORAL CHOLECYSTOGRAPHY:

It identifies the filling defects in a visualised, opacified gallbladder after administering oral radiopaque compound that passes into the gallbladder.

It is contraindicated in patients with –

- ✓ vomiting
- ✓ biliary obstruction
- ✓ Jaundice
- ✓ Hepatic failure.

iv. COMPUTED TOMOGRAPHY:

CT has a sensitivity of about 55% - 65% in identifying the gallstones.[21, 32, 40] This is because of isodense nature of both gallstone and bile, and stones are identified only if they are calcified.

v. SCINTIGRAPHY:

It is useful to visualize the biliary tree, assess liver and gallbladder function. Non-visualization of gallbladder at 2 hours after injections is the evidence of cystic duct obstruction.[41, 42] Biliary dyskinesia can be documented by biliary scintigraphy followed by CCK administration, when gallbladder contraction accompanies colic pain in patients without evidence of stones.

vi. INTRAOPERATIVE CHOLANGIOGRAPHY:

The first operative cholangiogram was done by Micken in 1936. Mirizzi in 1937 performed the first cystic duct cholangiography. In 1991, Reddick and Oslen published the 1st description of laparoscopic guided cholangiography.

TECHNIQUES:

- ✓ Cystic duct cholangiography.
- ✓ Gallbladder cholangiography.
- ✓ Kumar's technique.

INDICATIONS FOR ROUTINE IOC:

- Detection of unsuspected CBD stones
- To detect anomalous anatomy
- Presence of accessory duct
- Short cystic duct
- Identification of iatrogenic injury

COMPLICATIONS OF GALLSTONES:

1) Acute cholecystitis
2) Chronic calculus cholecystitis
3) Gallstone pancreatitis
4) Gallstone ileus
5) Choledocholithiasis with or without cholangitis
6) Gallbladder carcinoma

MANAGEMENT OF CHOLELITHIASIS:

In early times, dissolution of gallstones was the most fascinated management by physician.

In 1782, Durande gave the idea of dissolving gallstones.

In 1975, Makino used **urso-deoxycholic acid** for gallstones dissolution.

Management of cholelithiasis can be done by:

- ✓ Medical management
- ✓ Open cholecystectomy
- ✓ Laparoscopic cholecystectomy
- ✓ Extracorporeal shock wave lithotripsy (ESWL)

MEDICAL MANAGEMENT

Ursodiol (ursodeoxy cholic acid) is used in medical management, which constitutes less than 5% of total bile salt pool.

PHARMACOKINETICS:

After oral administration, when it gets absorbed, it gets conjugated with glycine or taurine and excreted in bile. This conjugated ursodiol undergoes enterohepatic circulation. The half-life is approximately 100 hrs.

On long term administration, a small amount of unabsorbed unconjugated or conjugated ursodiol passes into colon where it is either excreted or undergoes de-hydroxylation by bacteria to lithocholic acid, which is a potential hepatic toxic substance.

PHARMACODYNAMICS

1) Decreases the cholesterol content of bile by reducing hepatic cholesterol secretion.

2) Stabilizes hepatocyte canalicular membranes, through a reduction in concentration of endogenous bile acids or through inhibition of immune mediated hepatocyte destruction.

CLINICAL USES

1) Dissolution of small cholesterol gallstones. A dose of 10 mg/kg/day for 12-24m, dissolution occurs in up to 50% of patients with non calcified gallstones of size < 5-10 mm.
2) Prevention of gallstones in obese patient undergoing rapid weight loss.
3) At dose of 13-15 mg/kg/day is helpful for patients in:[44,45]
    - ✓ Early stage primary biliary cirrhosis
    - ✓ Reducing liver function abnormalities
    - ✓ Improving liver histology.

ADVERSE EFFECTS

It usually doesn't have serious adverse effects. Bile salt induced diarrhoea is uncommon.

OPEN CHOLECYSTECTOMY

PREOPERATIVE PREPARATION

1) Blood coagulation should be normalised by giving vitamin K (I.M. in 3 doses).
2) A prophylactic antibiotic, 2nd generation cephalosporin is appropriate.

3) Subcutaneous heparin or anti-embolic stocking are used to prevent deep vein thrombosis.

INDICATIONS FOR OPEN CHOLECYSTECTOMY:

1) Poor pulmonary or cardiac reserve
2) Suspected or known gallbladder cancer
3) Cirrhosis and portal hypertension
4) Third trimester pregnancy
5) Combined procedure
6) Conversion from laparoscopic approach

PROCEDURE:

KOCHER'S INCISION- a short right upper transverse incision is made centered over the lateral border of the rectus muscle. The gallbladder is appropriately exposed and packs placed on the hepatic flexure of the colon, the duodenum and the lesser omentum. A retractor is used to keep the pack in position. A duval forceps is placed on the infundibulum of the gallbladder and the peritoneum duct and the cystic artery. The cystic artery is ligated and cut. Then cystic duct is ligated and divided. Drain is placed before closure.

LAPAROSCOPIC CHOLECYSTECTOMY

It has become the most common mode of treatment replacing open cholecystectomy. In1992, the National Institute of Health (NIH) consensus

development conference stated that laparoscopic cholecystectomy "provides a safe and effective treatment for most patients with symptomatic gallstones."[47, 48]

Since its introduction, the number of cholecystectomy performed in US has increased from 5 lakh per year to 7 lakh per year.[11, 45, 49]

INDICATIONS OF LAPAROSCOPIC CHOLECYSTECTOMY

1) **Symptomatic cholelithiasis**
    a) Acute cholecystitis
    b) Biliary colic
    c) Gallstone pancreatitis

2) **Asymptomatic cholelithiasis**

    Patient with asymptomatic calculi have <20% chance of ever developing symptoms, and the risk associated with the prophylactic operation outweigh the potential benefit of surgery in most patient. Hence prophylactic surgery is recommended in:

    a) Sickle cell disease: such patients have hepatic and vaso-occlusive crisis that can be difficult to differentiate from cholelithiasis.[50,51]
    b) Total parenteral nutrition
    c) Chronic immune-suppression.
    d) No quick access to medical care.

e) Patient undergoing procedures for other indications.

f) Biliary dyskinesia or acalculous cholecystitis

g) Gallbladder polyps >1 cm in diameter

h) Porcelain gallbladder

CONTRAINDICATIONS TO LAPAROSCOPIC CHOLECYSTECTOMY:

ABSOLUTE

1) Unable to tolerate general anaesthesia
2) Suspicion of carcinoma
3) Refractory coagulopathy

RELATIVE

1) Cholangitis
2) Previous upper abdominal surgeries
3) Diffuse peritonitis with hemodynamic compromise
4) Cirrhosis and / or portal hypertension
5) Cholecysto-enteric fistula
6) Morbid obesity was a contraindication previously due to shorter length of trochar which caused problem in creating pneumoperitoneum
7) Chronic obstructive pulmonary disease
8) Pregnancy: due to unknown effect of co_2 on foetus hence avoided in first trimester.

PATIENTS LIKELY TO REQUIRE CONVERSION:

a) **Multiple prior operations-** due to difficulty in safe access to peritoneal cavity.
b) **Acute severe cholecystitis:** due to difficult dissection secondary to inflammation, adhesions or edema.
c) **Acute pancreatitis:** difficult visualization of Calot's triangle due to oedematous pancreatic head.
d) **Abnormal anatomy:** greater risk of biliary / vascular injury.
e) **Cirrhotic liver:** higher chances of liver injury and haemorrhage.
f) **Third trimester pregnancy:** higher risk of injury to uterus during access.
g) **Morbid obesity:** causes difficulty in access and dissection.
h) **Evidence of generalised peritonitis.**
i) **Septic shock:** from cholangitis.

APPROACHES:

A. NORTH AMERICAN APPROACH:

The patient is kept in supine anti-trendlenberg position (15 degree head up tilt) with left lateral tilt (15-20 degree).[23, 26, 52] By this position the bowel and omentum falls down and medially, away from the operative site.

PORT PLACEMENT:

Ports are placed by screwing motion by using second hand to prevent in advertent plunge of trocar. A counter traction should be provided on the abdominal wall while placing the first trocar.

- 10 mm port is placed in midline usually through **umbilical incision**.
- **Sub-umbilical** position is preferred in:
    - ✓ Cirrhosis due to presence of dilated, tortuous anastomotic veins in periumbilical region.
    - ✓ Visceroptic liver
    - ✓ Hepatomegaly
    - ✓ Pendulous abdomen

If previous abdominal surgery has been performed through a vertical midline incision, then abdomen is insufflated through a site adjacent to the umbilicus, and a primary 5 mm trocar is placed in the right upper quadrant. The 10mm trocar is placed under direct vision, avoiding the adhesions of previous operation, under direct vision through a 5 mm telescope passed through 5mm port. Pneumoperitoneum is created by HASSON.[27, 53, 53]

TECHNIQUE:

Another 10 mm port is placed in the epigastrium starting from the midline and angling toward the gallbladder, at the level of the inferior edge of the liver and to the right of the falciform ligament.

A 5mm trocar is placed 2 to 3 cm below the coastal margin in mid-clavicular line. The fourth, a 5 mm trocar is generally placed in the anterior axillary line, several centimeters below the fundus of gallbladder, but its position is variable.

B. FRENCH / EUROPIAN APPROACH:

The patient in semi lithotomy position with leg in allen stirrups such that the thighs almost parallel to the ground. Operating surgeon stands in between the legs of the patient, with camera surgeon on right and assistant on left.

PORT PLACEMENT:

The camera port is placed at the umbilicus; 5mm epigastric port is placed to allow the retraction by assistant. 10 mm right hand working port is placed in left hypochondrium or in the midline between the camera port and the epigastric port and the left hand working port (5mm) is placed in the right hypochondrium.

ADDITIONAL PORT:

a) **Left lumbar 5 or 10 mm port:** for three prong or flat blade retractor for downward traction of colon, omentum and duodenum. This procedure gives wide exposure of the hilum.
b) **5 mm port midway between epigastric and right mid-clavicular ports:** for lifting the quadrate lobe using blunt tipped retractors (French technique) e.g. in liver cirrhosis, left lobe gallbladder.

PNEUMOPERITONIUM

Laparoscopic cholecystectomy is performed with a carbon dioxide pneumoperitoneum at a pressure of 15 mm of Hg pressure. Other gases like helium, nitrogen oxide and argon is also being used.

TECHNIQUES:

A. VERESS NEEDLE TECHNIQUE:

Pneumoperitoneum is generally created by sliding a veress needle through the umbilicus. After confirming its position by pushing saline through the needle from a plunger less syringe, and then attaching the needle to tubing from carbon dioxide.

Initially the flow rate of CO_2 is kept below 21/ min to insure the proper placement. Confirmation of intra-abdominal position of the needle can be obtained by observing for:

- ✓ uniform abdominal distention
- ✓ tympany
- ✓ ability to vary the intra abdominal pressure

Initial pressure greater than 10 mm Hg nearly always reflect retroperitoneal placement of the needle. Once its position is confirmed, the flow rate can be increased until 15 mm Hg of pressure is attained.

B. OPEN (HASSON) LAPAROSCOPY TECHNIQUE:

Here abdominal cavity is entered under direct vision. Once the cavity is entered, initial trocar is inserted and its position is secured with 2 stay sutures. The abdominal cavity is then insufflated with carbon dioxide.

PATIENT PREPARATION, EQUIPMENT AND ANAESTHESIA:

Ideally, the patient is placed on fluoroscopy table with the table turned backward for easy access to mid abdomen by c-arm.

EQUIPMENT

a) High quality video scope.

b) 300 w light source.

c) Two high resolution monitors.

d) High-flow carbon dioxide insufflator.

e) Four trocars: 2-10 mm trocars and 2-5 mm trocars.

f) Hand instruments :

- ✓ Mono-polar electrode c-hook with suction and irrigation
- ✓ A fine tipped dissector
- ✓ 2 gallbladder grasper
- ✓ A large gallbladder extractor
- ✓ A pair of scissors
- ✓ Medium to large hemoclip applier.

g) 10 mm stone retrieval grasper

h) Micro scissors

i) Specialized cholangiogram clamp

j) 4-5 mm French catheter to perform cholangiogram.

ANAESTHESIA TECHNIQUE

Nitric oxide is generally avoided to minimize the likelihood of bowel distension. Intravenous fluids must be rushed, to minimize the loss of fluid through closed abdomen and pneumoperitoneum is a strong stimulator of anti-diuretic hormone. End tidal pCO_2 is monitored to check for hypercarbia and acidosis secondary to carbon dioxide pneumoperitoneum.

Narcotics used in smaller doses. Antiemetic is used to lessen post operative nausea. Once the patient is anaesthetised and intubated, a foley catheter, sequential compression devices and orogastric tube are generally placed.

A. <u>EXPOSURE OF PORTA HEPATIS:</u>

The fundus of the gallbladder is held with a ratchet grasper and retracted in cranial direction. This lifts the right lobe of the liver and exposes the calots triangle and hilum of the liver. The adhesions are carefully taken down beginning near the fundus and proceeding down towards the neck.

B. DISSECTION OF THE CHOLECYSTEOHEPATIC TRIANGLE (CALOTS TRIANGLE)

In tensely distended GB, it may be decompressed by two ways-

- ✓ Verees needle aspiration
- ✓ Direct introduction of mid-clavicular trocar into the fundus of gallbladder and aspiration.

An atraumatic (dolphin- nosed) non looking grasper is introduced through the left hand working port to hold the infundibulum and retract it downwards and right. Using Maryland's forceps introduced through the epigastric port, the infundibulum is held and breached by using small bursts of cautery current. Peritoneum on anterior and posterior aspect is stripped down.

The infundibular grasper is moved infrolaterally and superomedially (flag technique) to aid the dissection of anterior and posterior surface of Calot's triangle.

C. IDENTIFICATION OF THE CYSTIC DUCT AND ARTERY:

Methods for ductal identification in laparoscopic cholecystectomy are-

i. **Infundibular or infundibular-cyst technique:** here cystic duct is isolated by dissection on the front and the back of the Calot's triangle and from here it is traced on to the gallbladder. This is referred to as seeing a

funnel shape i.e. the gallbladder is seen to funnel down to terminate into the cystic duct.

ii. **Critical view of safety triangle:** here complete dissection of the cholecystohepatic triangle and separation of the base of the gallbladder infundibulum from the liver bed. After this, the two structures entering the gallbladder are cystic duct and artery. Cystic duct is identified at the junction of gallbladder (SAFETY ZONE) and followed down for adequate length for cholangiography. It is not necessary to identify and dissect the cystic duct CBD junction (DANGER ZONE).The cystic artery is identified along with its anterior and posterior branches by blunt dissection. Both cystic duct and artery are clipped, 2 clips on the cystic duct side and 1 on the gallbladder side. Before clipping the cystic duct the stones in the cystic duct are milked back to GB.

Artery is divided before the duct, and in certain cases duct is divided first.

D. <u>DETACHMENT OF GALLBLADDER FROM THE LIVER BED:</u>

The GB can be detached from the liver bed using a spatula with monopolar cautery, hook with monopolar cautery, scissors with monopolar cautery or harmonic scalpel. Traction and counter traction facilitate dissection. Any inadvertent spillage of bile or stones from the GB during the procedure should be immediately controlled by applying clips, pre-tied loops or reapplying the grasping clamp. Before complete dissection of GB, the liver bed is inspected for

adequate hemostasis or bile leak. The cystic duct remnant and cystic artery stumps are examined. After complete hemostasis is achieved GB is separated completely.

E. EXTRACTION OF GALLBLADDER:

It can be done through umbilical or epigastric port. Epigastric port is preferred because:

i. No need of changing camera port
ii. Facilitates thorough rinsing to avoid port traction infection.
iii. By extending skin incision, the facial opening can be easily dilated and majority of GB extracted.
iv. Fascial opening closed easily by cutaneous approach.
v. Better cosmetic appearance.

A claw shaped gallbladder extraction forceps is introduced and used to grasp the neck of gallbladder. If the GB is too distended, the neck is pulled out through the skin incision, small nick made and bile suctioned and stones crushed using sponge holder. If the GB is thick preventing its extraction, the fascial incision is enlarged using a closed Robert's clamp or extending it. Infected GB or GB with suspicion of carcinoma is placed in a sterile bag before extraction to reduce the port site infection.

F. FINAL INSPECTION AND IRRIGATION:

After GB extraction, the epigastric port is replaced and surgical site inspected for bleeding. A thorough wash is given to GB bed, Morrison's pouch, para-colic gutter and peri-hepatic areas with saline. Venous ooze is controlled from the liver bed by:

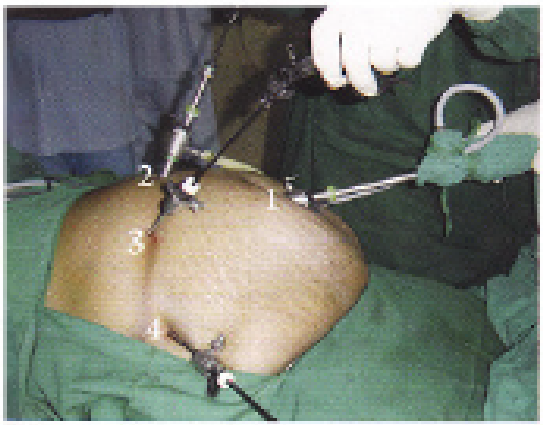
i. Gelatin sponge soaked in hemostatic solution eg. Hemlock solution.
ii. Use of harmonic ball application.
iii. Rarely intracorporeal suturing.
iv. Argon plasma coagulator.

G. DRAINAGE AND CLOSURE:

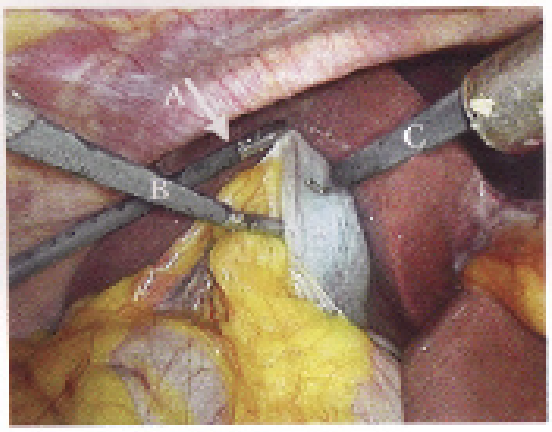
If drain is required, a 14 F Redivac tube is placed through 5mm trocar site lateral most port. Trocars are removed under direct vision to check for bleeding from trocar site. Pneumoperitoneum evacuated and 10 mm ports closed with vicryl subcuticular stitch/ skin clip/ dermabond.

Fig.17 Steps of Laparoscopic Cholecystectomy

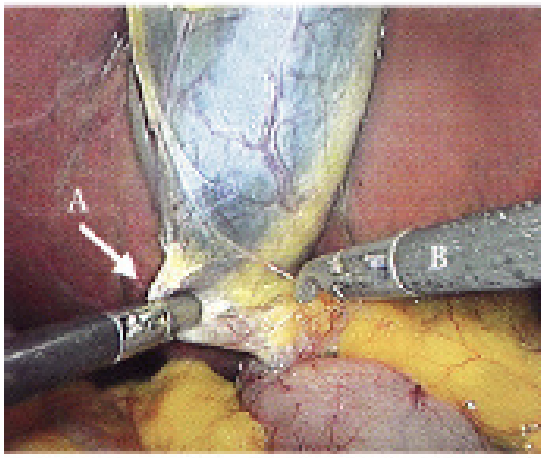
STEP 1: Patient Position



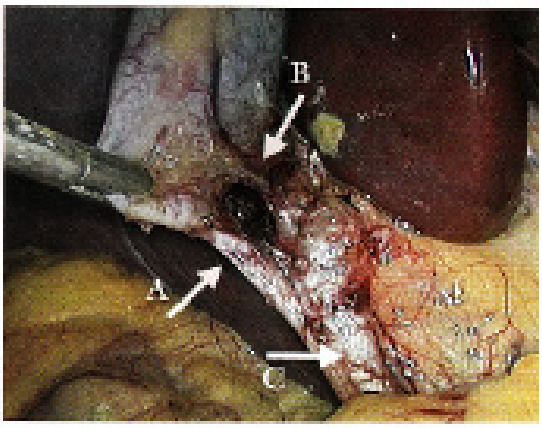
STEP 2: Port Placement



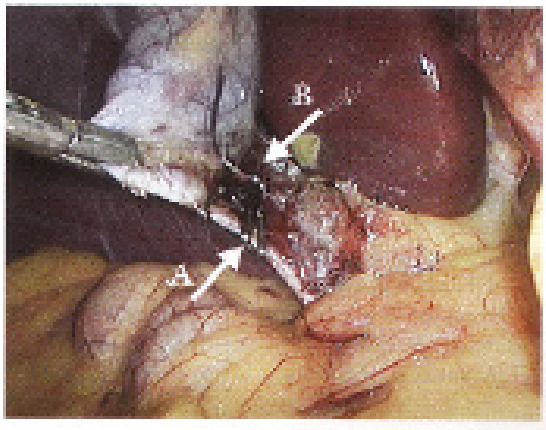
STEP 3: Exposure of Porta Hepatis



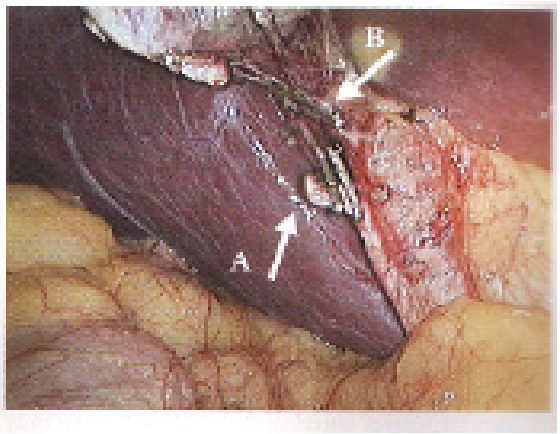
STEP 4 : Dissection of Calot's Triangle



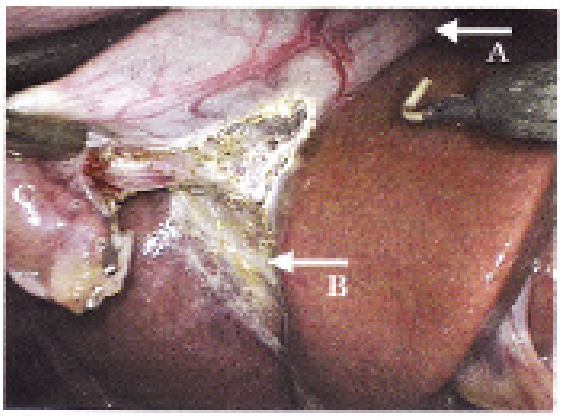
STEP 5: Identification of Cystic Duct(A), Cystic Artery(B) and CBD(C)



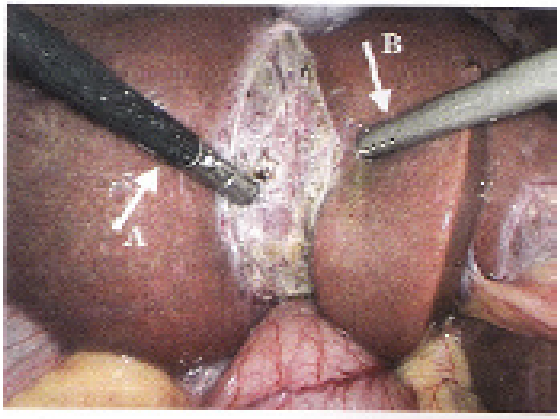
STEP 6: Clipping of Cystic artery and Cystic duct



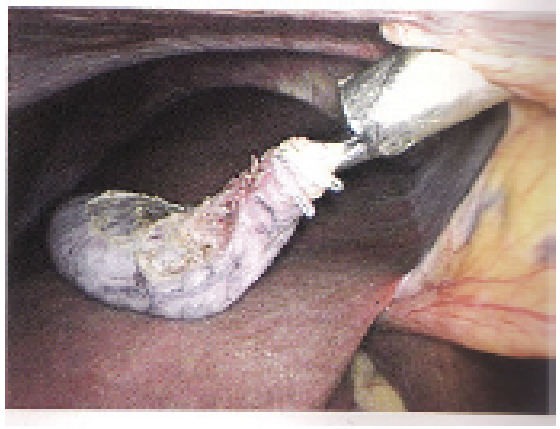
STEP 7: Division of Cystic artery and Cystic duct



STEP 8: Detachment of GB from the Liver bed



STEP 9: Inspection of Liver bed



STEP 10: Etraction of GB

STEP 11: Extraction of the entire unit

COMPLICATION:

A) HEMORRHAGE

i) TROCAR SITE BLEEDING

Trocar site bleeding can be prevented by control of bleeding following skin incision and before inserting trocar. Any subcutaneous vessel in subcutaneous tissue should be avoided during insertion. Detection: the blood may run down the abdominal wall or drip down the instruments into the operative field. Management: pressure over the site of bleeding by tilting the trocar. Injection of epinephrine 1:10000 in the vicinity of the bleeding site. Screwing in the anchoring device of a disposable trocar may compress and stop the bleeding.

ii) HEMORRHAGE DUE TO BLUNT DISSECTION OF ADHESIONS can be managed with electrocautery.

iii) SUDDEN AND PULSATILE BLEEDING IN CALOT'S TRIANGLE

Bleeding in the Calot's triangle can be prevented by careful dissection and proper application of clip to cystic artery. Management: Retraction of the GB is released and the GB is gently pushed into the Calot's triangle to obtain temporary respite during which additional port is placed between the umbilical and the epigastric ports.by repeated suction and irrigation, the blood is cleared from the operative field and the bleeding vessel is precisely identified and clipped.

iv) GALLBLADDER FOSSA BLEEDING:

GB fossa bleeding can be controlled by electro-cautery, packing the site with hemlock soaked gel foam, figure of eight stitch in case of spurter from liver parenchyma.

v) PERFORATION OF GB

GB perforation seen in acute cholecystitis and while detaching GB from the liver bed. This can be prevented by confining to the areolar tissue between the GB and the liver bed during dissection and decompression of the gall bladder if distended.

TABLE 2: Complications secondary to gallstone spillage

INFECTIVE	CUTANEOUS	MECHANICAL
Liver abscess	Sinus	Intestinal obstruction
Retrohepatic abscess	Port tract infection	
Subhepatic abscess	Granuloma formation	
Retroperitoneal abscess	Colocutaneous fistula	
Loin abscess		
Pelvic abscess		

Management**:** Copious irrigation and suction will remove majority of small stones while larger ones are removed using laparoscopic tissue pouch. Drainage catheter is placed. Perforated site must be closed with pretied ligature or by holding with the grasper.

vi) DIFFICULTY IN EXTRACTION OF THE GALLBLADDER

It is seen in gallbladder containing large stones and those with thick wall. In GB containing large stones, the GB is placed in an endobag, the neck retrieved out through the abdomen and stones are crushed and removed. In GB with thickened wall, the GB is placed in an endobag and extracted.

vii) OCCULT CARCINOMA

In cases suspected to have carcinoma intra-operatively, frozen section is sent and if frozen section is positive for carcinoma, then conversion to open technique is considered and radical surgery with excision of port sites done.

viii) POST OPERATIVE BILE LEAK

Post operative bile leak occurs due to inadvertent injury to the CBD, the right hepatic duct or accessory bile duct. In case of acute inflammation, the clip applied to the cystic duct may become loose once the edema subsides and subsequently slip off. This can be prevented by accurate identification of the cystic artery and duct, minimum use of cautery in Calot's triangle dissection and appropriate choice of laparoscopic subtotal cholecystectomy. In case of acute cholecystitis, when correct application of the clip doubtful, it may be advisable to use a intra-corporeal suturing or pre-tied suture loop to occlude the cystic duct. Postoperative bile leak should be suspected in patients with fever, tachycardia and upper abdominal pain and tenderness persisting or appearing unexpectedly. The diagnosis can be confirmed by USG or ERCP. If drain is placed most of the minor leak will heal with expectant management. In some

persistent cases, it may be advisable to decrease the intraductal pressure by nasobiliary drainage, endoscopic spincterotomy or transpapillary stenting.

ix) BILE DUCT INJURY

Incidence of CBD injury during LC exceeds that of open cholecystectomy i.e. 0.5% s 0.2%.[21] Reasons for the increase in injury during LC included loss of hepatic information, incorrect traction forces to the gallbladder, and injudicious use of cautery inside of the triangle of Calot. Risk factors that increase the risk of CBD injury include acute cholecystitis, aberrant anatomy. The most common anatomic variant is an aberrant right hepatic duct.

PREVENTION

i) Use a 30 degree laparoscope and high-quality imaging equipment.

ii) Apply firm cephalic traction to the fundus and lateral traction to the infundibulum so that the cystic duct is perpendicular to the CBD.

iii) Dissect the cystic duct where it joins the gallbladder.

iv) Expose the “critical view of safety” prior to dividing the cystic duct.18

v) Convert to open procedure if the infundibulum cannot be mobilized or bleeding or inflammation obscures the triangle of Calot.

vi) Perform routine intraoperative cholangiography. A recent study using an American Medicare database found a reduction in CBD injuries with routine use of IOC (from 0.58% to 0.39%).

CLASSIFICATION

The Stewart-Way classification is derived from analysis of a series of LC associated CBD injuries.

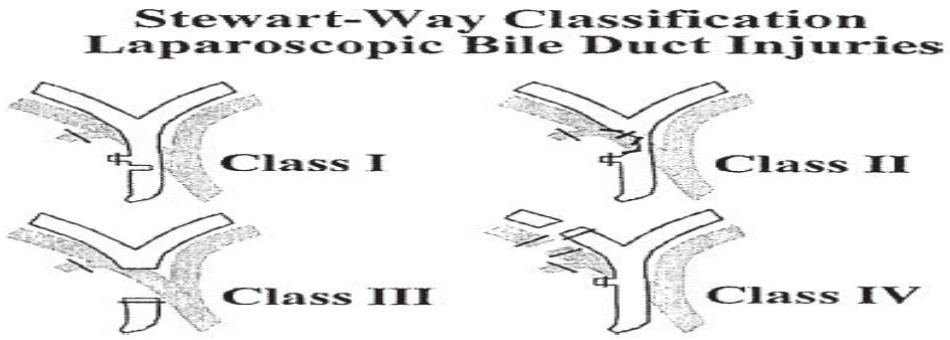


FIG 18: Stewart- Way classification of bile duct injury

TABLE 3 STEWART-WAY CLASSIFICATION

Class I	Incision in cystic duct extended into CBD CBD mistaken for cystic duct
Class II	Lateral damage to the CHD from cautery or clips placed on duct Associated bleeding and poor visibility
Class III	CBD mistaken for cystic duct, not recognized CBD, CHD, Rt, Lt hepatic ducts transected and /or resected
Class IV	RHD mistaken for cystic duct, RHA mistaken for cystic artery, RHD and RHA transected Lateral damage to the RHD from cautery or clips placed on duct.

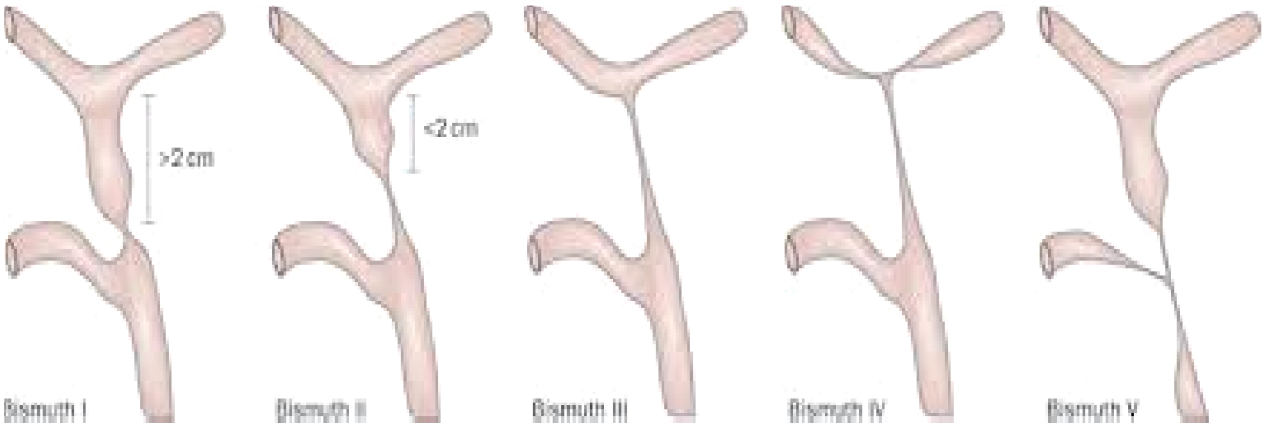


Fig.4: Based on the level of CBD injury (BISMUTH CLASSIFICATION)

Table.4. Bismuth Classificaation

Type I	Low common bile duct; stump >2 cm
Type II	Middle common hepatic duct; stump <2 cm
Type III	Hilar-confluence of right and left ducts intact
Type IV	Right and left ducts separated
Type V	Involvement of the intrahepatic ducts

It is managed by biliary enteric anastomosis. This is to prevent cholangitis and biliary strictures.

g) BOWEL INJURY

Injury to bowel can occur during trocar insertion or dissection in the right upper quadrant, especially when using electrosurgical devices. The jejunum, ileum and colon can be injured by veress needle and trocars while duodenum is likely to be injured during dissection. Any structure fixed to the under surface of the umbilicus like the urachus or a meckel's diverticulum is more susceptible to injury during access. The rate of bowel injury between 0 and 0.4% has been reported in various studies.[22] Deziel et al carried out retrospective analysis and found that mortality rate following all bowel injuries during laparoscopic cholecystectomy was 4.6% while it was 8.3% for duodenal injuries.22 Incidence of hollow viscus injury following closed veress needle technique and open access for pneumoperitoneum are the same.

h) WOUND INFECTION AND INCISIONAL HERNIA

The risk of incisional hernia is 0.5% and the risk of wound infection following laparoscopic cholecystectomy is less than 1% and [23] Usage of a retrieval bag for extraction of GB and closure of all port sites larger than 8mm may avoid these complications.

i) DIAPHRAGMATIC INJURY

Diaphragmatic injury may be due to either cautery or by mechanical puncture by an instrument while retracting the fundus cranially with excessive force.[24, 25]

j) PANCREATITIS

h) PNEUMOPERITONEUM RELATED COMPLICATIONS

Pneumoperitoneum related complications include carbon dioxide embolism, Vasovagal reflex, cardiac arrhythmias and hypercapnia acidosis. Hypercapnia and acidosis are due to absorption of carbon dioxide from the peritoneal cavity. Sudden increases in Paco2 may be related to port slippage and extraperitoneal or subcutaneous diffusion of co2. It is managed by desufflating the abdomen for 10 to 15 min. If re-insufflation results in recurrent hypercapnia, then change the insufflations gas or convert to open. Carbon dioxide embolism is characterized by unexplained hypotension and hypoxia. Characteristic millwheel murmur is detected on auscultation. This is produced due to the contraction of right ventricle against the blood gas interface. There is an exponential decrease in end tidal co2 due to complete right ventricular outflow

obstruction. It is managed by immediate evacuation of pneumoperitoneum and placement of the patient in left lateral decubitus, head down (Durant) position. This allows the CO2 bubble to float to the apex of the right ventricle, where it is less likely to cause right ventricular outflow obstruction. Patient is hyperventilated with 100% oxygen.

ADVANTAGES AND DISADVANTAGES OF LC COMPARED TO OC

TABLE 5: Advantages and disadvantages of lc compared to oc

ADVANTAGES	DISADVANTAGES
Less post operative pain	Lack of depth perception
Smaller incision	View controlled by camera operator
Better cosmesis	More difficult to control hemorrhage
Shorter hospitalization	Decreased tactile discrimination (haptics)
Earlier return to full activity	Potential co2 insufflation complications
Decreased total costs	Adhesions/inflammation limit use
	Slight increase in bile duct injury

CONVERSION

In 5-10% of cases, conversion to open cholecystectomy may be needed for safe removal of gallbladder; the risk factors for conversion were male sex, obesity, cholecystitis and choledocholithiasis.[26]

RISK FACTORS OF DIFFICULT LAPAROSCOPIC

CHOLECYSTECTOMY

i) CLINICAL RISK FACTORS

a) Stocky male patients due to difficulty in initial port placement3[4, 35]

b) Multi-parous women with flabby abdomen due to thinned out lower abdominal musculature the effect of pneumoperitoneum is only in the lower abdomen. Hence there is less space in right hypochondrium to work.

c) Previous upper abdominal surgery[36]

d) Cirrhosis of liver

e) Present or previous acute cholecystitis or acute severe pancreatitis[37]

f) Previous treatment: percutaneous drainage or cholecystostomy

II) ULTRASOUND CRITERIAS

a. Thick walled gallbladder (>4 mm) [38]

b. Contracted (non-functioning) gallbladder

c. Packed stones and large calcified GB.

d. Polyp or mass lesion without acoustic shadow

e. Evidence of acute cholecystitis:-impacted stones

i. Edematous gallbladder wall

ii. Pericholecystic fluid collection

iii. Air in the gallbladder (emphysematous cholecystitis)

iv. Subphrenic collection

v. Intraperitoneal fluid collection due to perforated GB

f. Fatty liver with hepatomegaly

g. Cirrhosis of liver

h. Portal vein thrombosis with cavernoma

SAFETY MEASURES

a) Selective open technique of pneumoperitoneum

b) Intraoperative cholangiography to identify biliary anatomy and the CBD stones.

c) Laparoscopic ultrasound is useful in mapping biliary and vascular anatomy and is superior to operative cholangiogram.

d) Adequate instrumentation:

i) Toothed graspers to grasp and retract thick walled gallbladder.

ii) Specialized needle drivers and holders

iii) Five pronged retractors.

e) Hydrodissection

f) Preliminary decompression

g) Additional ports for retraction to get adequate exposure

h) Caudal traction of the hepatoduodenal ligament using multipronged retractor. The port is placed in the left midclavicular line, midway between the camera port and the epigastric port.

i) Dipping retractor for quadrate lobe lifting (French technique)

PROBLEMS IN DIFFICULT CHOLECYSTECTOMY

ACCESS PROBLEMS

a) ADHESIONS

Post-operative adhesions: In lower abdominal scars, the veress needle is inserted at the site of proposed epigastric port. The umbilical port is inserted under visual guidance. In open appendicectomy scar, Hasson method is the ideal technique for creating pneumoperitoneum. In case of upper abdominal scars present in the midline or right Para median position, the left subcostal veress needle insertion (palmer's point) is used to create pneumoperitoneum. Conversion rate as high as 25% has been reported in patients with extensive upper abdominal adhesions.[28]

Inflammatory adhesions: is usually due to acute cholecystitis or acute severe pancreatitis. These adhesions can easily be removed using suction nozzle. But if the adhesions are organized then sharp dissection is done.

b) INCISIONAL HERNIA

In cases of lower abdominal incisional hernias, appropriate repair could be accomplished after completing laparoscopic cholecystectomy either by open or laparoscopic technique.

c) OBESITY

The veress needle insertion and the insertion of first trocar is difficult. Cystic artery and cystic duct are covered with thick fat hence dissection is difficult.

d) CIRRHOSIS

Due to adhesions with increased vascularity, difficult traction of liver, inadequate exposure of hilum, high risk of GB bleed and high risk hilum.

CONCOMITANT PATHOLOGY

a) MUCOCOELE

Mucocoele is difficult to retract and apply grasping forceps. It is managed by decompression of the GB, using toothed forceps for retraction of GB, removal of the impacted stone either by dislodging into the GB or through an incision over the cystic duct after applying distal clip.

b) GANGRENOUS GB

Due to difficulty in grasping, loss of tissue plane, difficulty in exposure of Calot's triangle, performance of intraoperative cholangiogram is difficult, spillage of stones and infected bile; gangrenous GB is difficult to operate.

c) EMPYEMA

d) SCLEROATROPIC GB

The GB is contracted, fibrosed and densely covered with extensive adhesions. Adhesions of the duodenum and the colon are very common and access to Calot's triangle is difficult due to fibrous scarring.

e) MIRRIZZI'S SYNDROME

LC is difficult in Mirrizzi's syndrome due to contracted GB with extensive adhesions, CBD may be mistaken for cystic duct and chances of CBD injuries are more and if fistula is not recognized during surgery, biliary

peritonitis may occur. Preoperative ERCP is done in all cases to assess the pathological nature and anatomy of the biliary system.

f) PORCELAIN GB

The prevalence of porcelain GB in cholecystectomy specimen ranges from 0.06% to 0.8%.[30] Decompression of the gallbladder and traction is difficult due to calcified wall. Toothed forceps can be used for cranial traction of the GB. Calcification of the cystic duct may require endo-suturing or application of endoloops to the cystic duct.

g) CHOLECYSTOENTERIC FISTULAS

Cholecystoenteric fistula is an incidental finding in 0.5 to 0.7% of cases of laparoscopic cholecystectomy for biliary disease.[31] The diagnosis suspected by the presence of air in GB. Problems arise due to difficulty in identification of the anatomy, difficulty in performing cholangiography and due to the requirement of intra-corporeal suturing for closure of perforation.

h) ACUTE BILIARY PANCREATITIS

Difficulty in performing LC in acute biliary pancreatitis is due to- extensive adhesions, inflammatory phlegmon at the head of pancreas, edematous cystic duct and hepatoduodenal ligament, presence of ascites, pseudocyst pancreas in retro gastric position.

NEWER APPROACHES IN LAPAROSCOPIC CHOLECYSTECTOMY[65]

a) GASLESS LAPAROSCOPIC CHOLECYSTECTOMY:

Gasless LC is especially useful in patients with cardiorespiratory problems.

Here the abdominal wall is lifted mechanically allowing an adequate space for laparoscopic surgery.

b) SILS (Single incision Laparoscopic Surgery) CHOLECYSTECTOMY.

c) MPSI (Multi-port Single incision) CHOLECYSTECTOMY: It is a cost effective surgery compared to SILS which needs a special device.

EXTRACORPOREAL SHOCK WAVE LITHOTRIPSY (ESWL)

It is being used since 1986, to fragment stones. For the success of ESWL the criteria was laid down in Munich study.

The criteria should be:

i. Cholesterol stones
ii. Less than 3 in number
iii. Less than 3cm

The recurrence rate is 5-7% at 12months and 15% at 24 months

MATERIALS AND METHODOLOGY

STUDY DESIGN:

- ✓ It is a prospective study. The research population include all patients with symptomatic choleliatiasis attending the General Surgery OPD in Coimbatore Medical College hospital.
- ✓ Sample size: 50 Patients
- ✓ Duration of study is 1 year (November 2012 to November 2013)

MARTERIALS:

- ✓ Study data collection formats
- ✓ Paper
- ✓ Pencil

PERSONNEL:

- ✓ Radiologist
- ✓ Surgeons of all Units in Department of General Surgery
- ✓ Nursing and Theatre staff

METHODOLOGY:

The method for the study included screening of patients who presented with upper abdominal pain, or vomiting or dyspepsia or jaundice. Such patients were studied in detail clinically and investigated as per the proforma detailed below. Routine haematological and biochemical investigations were done. LFT and PT-INR were done in all patients. Ultrasonogram of the abdomen is done

after a 12 hour fast. The patients confirmed by USG examination were evaluated with following factors: age, sex, h/o previous hospitalization, BMI wt (kg)/ ht (mt2), abdominal scar, supraumbilical or infraumbilical, sonographic findings- wall thickness, GB size, number of stones, mobility of stones, stone size.

All the patients were received symptomatic treatment and vitamin K for 3 days pre-operatively.

Following evaluation the patient will be subjected to laparoscopic cholecystectomy and time taken, biliary / stone spillage or conversion were noted. All the patients were operated by experienced surgeons.

Post operatively cases were followed up for any complication. S/R was done 8th post OP day. All cases were followed up for any recurrent symptoms.

INCLUSION CRITERIA:

The patients above 13 years of age, presenting with symptoms and signs of Cholelithiasis and diagnosed by USG examination in surgical ward of Coimbatore Medical College Hospital, Coimbatore.

EXCLUSION CRITERIA:

Patients below 13 years of age.

Patients with CBD calculus, raised ALP, dilated CBD, where CBD exploration was needed.

Patients with features of acute cholecystitis, obstructive jaundice.gall stone pancreatitis.

Patients refusing surgery.

Patients not willing for laparoscopic cholecystectomy.

Definition of variables

- ✓ Age is considered as a continuous variable.
- ✓ Body habitus is treated as a dichotomous variable 1.obese [body mass index >30 Kg/m2] versus 2. non-obese).[1]
- ✓ Previous abdominal surgery is classified as any intra-abdominal surgery versus none.
- ✓ The sub-costal angle is classified as narrow and wide, narrow sub-costal angle was defined as < 90 degrees.
- ✓ Acute calculous cholecystitis is defined as acute onset right hypochondrial pain, associated with gall bladder calculi and pericholecystitic fluid collection. [2]
- ✓ Acute gallstone pancreatitis was defined as cholelithiasis with a raised serum amylase to ten times its normal level at any time prior to surgery.

The abdominal ultrasonological examination is done to assess six parameters, with each parameter classified into two classes

- ✓ The GB was classified as contracted or distended. It was defined as distended if the transverse diameter was greater than five centimeters.[3]
- ✓ GB wall was deemed thickened if wall thickness > 3mm.
- ✓ The mobility of the stone is determined by scanning the patient in various decubitus positions.

- ✓ Number of stones in Gall bladder. (Multiple versus Solitary).
- ✓ The largest stones's diameter is recorded and classified into two groups (<1 cm versus >1 cm)

The outcomes included the following operative observations:

- ✓ duration of surgery (in minutes),
- ✓ bleeding during surgery,
- ✓ access to peritoneal cavity,
- ✓ GB bed dissection,
- ✓ difficult extraction,
- ✓ Conversion to OC.

Bleeding during surgery was graded as minimal, moderate or severe. Moderate bleeding is defined as bleeding leading to tachycardia of greater than 100/min without drop in blood pressure.[3] Severe bleeding is defined as bleeding leading to tachycardia of greater than 100/min with a greater than 10 mmHg drop in blood pressure. Duration of surgery included the time from insertion of the Veress' needle to closure of the trocar insertion site[4] and is evaluated as a dichotomous variable (<90 min versus >90 min. The operating surgeon was not aware of the preoperative US results and gave a opinion on LC difficulty at the end of the surgery in a two-level classification (easy, difficult)

The parameters and outcomes are analysed using SPSS statistical software.

RESULTS

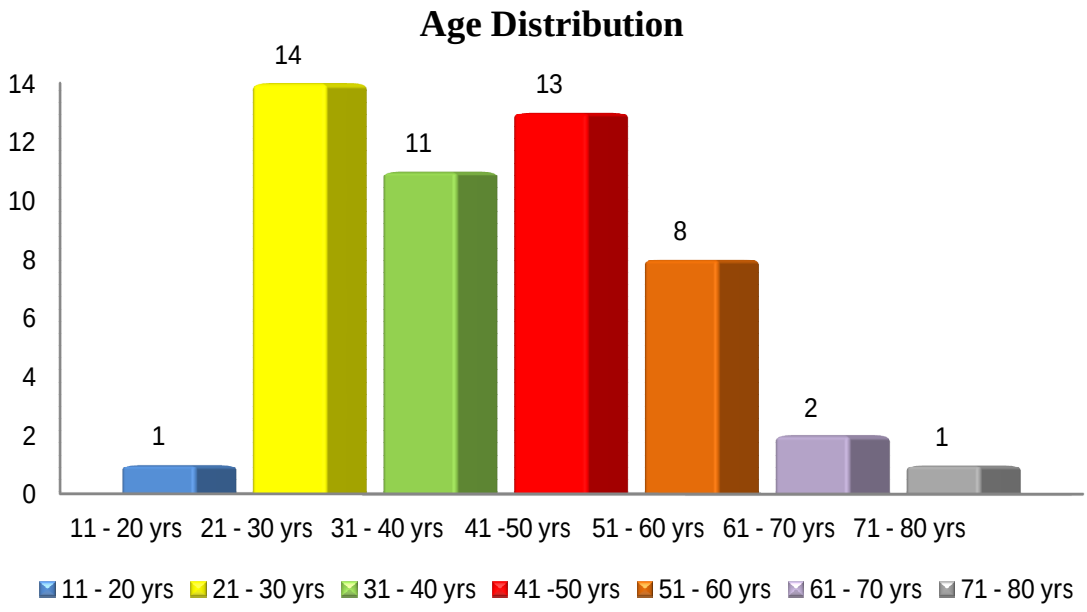
This study included 50 cases that were studied prospectively over a period of 12 months, from November 2012 to November 2013.

AGE DISTRIBUTION

In the present series the youngest patient was 15 yrs of age and the oldest was 75 yrs of age. Majority of the patients in the present series were in the age group of 31-40 yrs of age.

Table. 6 Age Distribution

AGE GROUP	NO. OF PERSONS	PERCENTAGE
11- 20 yrs	1	2%
21 - 30 yrs	11	22%
31 - 40 yrs	14	28%
41 -50 yrs	13	26%
51 - 60 yrs	8	16%
61 - 70 yrs	2	4%
71 - 80 yrs	1	2%
Total	50	100%

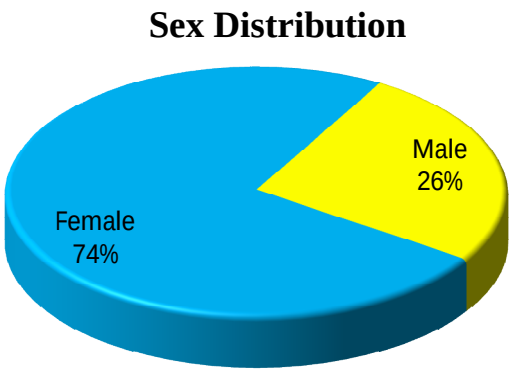


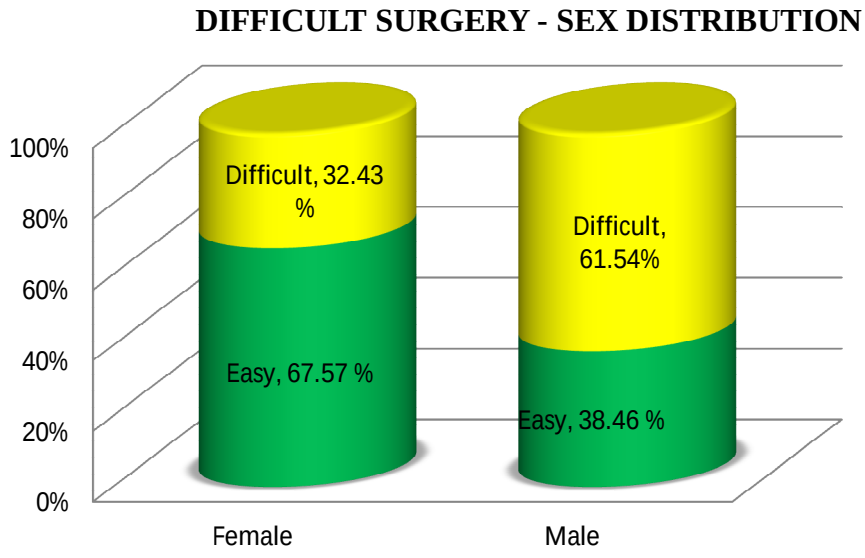
SEX DISTRIBUTION

Out of 50 patients 37 were females and 13 were male patients. The male:female ratio is 1:2.8.

Table.7 Sex distribution

Sex	Present Series	%	Hanif Seris	%
Male	13	26%	90	36%
Female	37	74%	160	64%
Total	50		250	





Sex Vs Difficult Surgery

Lap Chole Difficulty

Sex	0	1	
F	25 67.57 % 83.33 %	12 32.43 % 60.00 %	37 100.00 % 74.00 %
M	5 38.46 % 16.67 %	8 61.54 % 40.00 %	13 100.00 % 26.00 %
	30 60.00 % 100.00 %	20 40.00 % 100.00 %	50 100.00 % 100.00 %

Single Table Analysis

Odds- and Risk-based parameters

	Estimate	Lower	Upper
Odds ratio	3.3333	0.8973	12.3832
MLE Odds ratio (Mid-P)	3.2477	0.8664	13.1267
Fisher-Exact		0.7500	15.6096
Risk ratio	1.7568	0.8526	3.6198
Risk difference	29.1060	-1.3398	59.5519

Statistical Tests

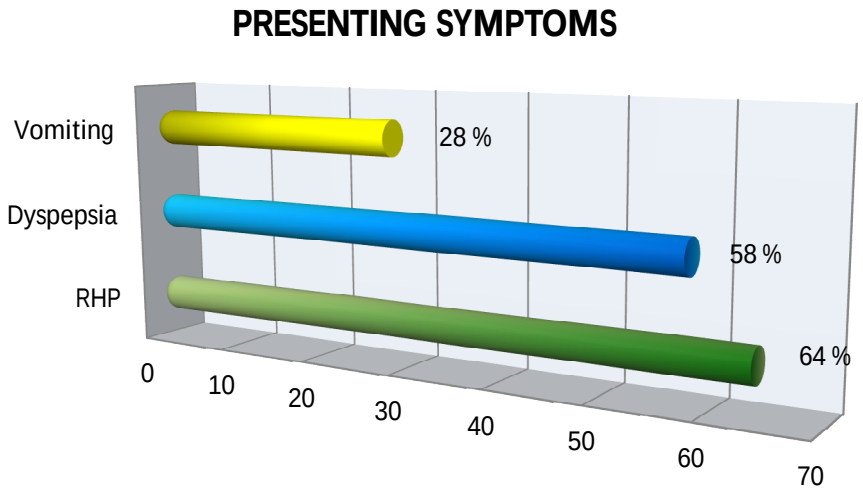
	X^2	2 Tailed P
Uncorrected	3.3957	0.0653675709
Mantel-Haenszel	3.3278	0.0681194648
Corrected	2.2912	0.1301075007

	1 Tailed P	2 Tailed P
Mid-P Exact	0.0405954228	
Fisher-Exact	0.0658891366	0.1003500324

Out of the 13 males involved in the study, 8 persons had difficult Cholecystectomy with four of the subjects having difficult bed dissection.

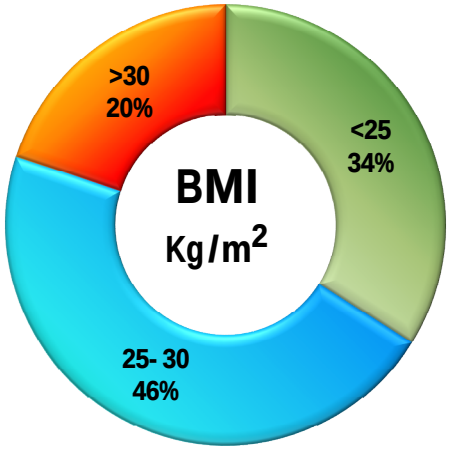
PRESENTING SYMPTOMS

Pain was the predominant symptom seen in all 50 patients. Right hypochondrial pain was present in 64% (32) of the patients, 58 % (29) of the patients had Dyspepsia, 28% (19) of the patients had vomiting.



BMI:

Of the 50 patients, 10 patients were obese, while 23 were overweight and 17 had normal BMI.



The following table depicts the influence of BMI > 30 kg/m^2 as a factor on the various steps of Laparoscopic Cholecystectomy.

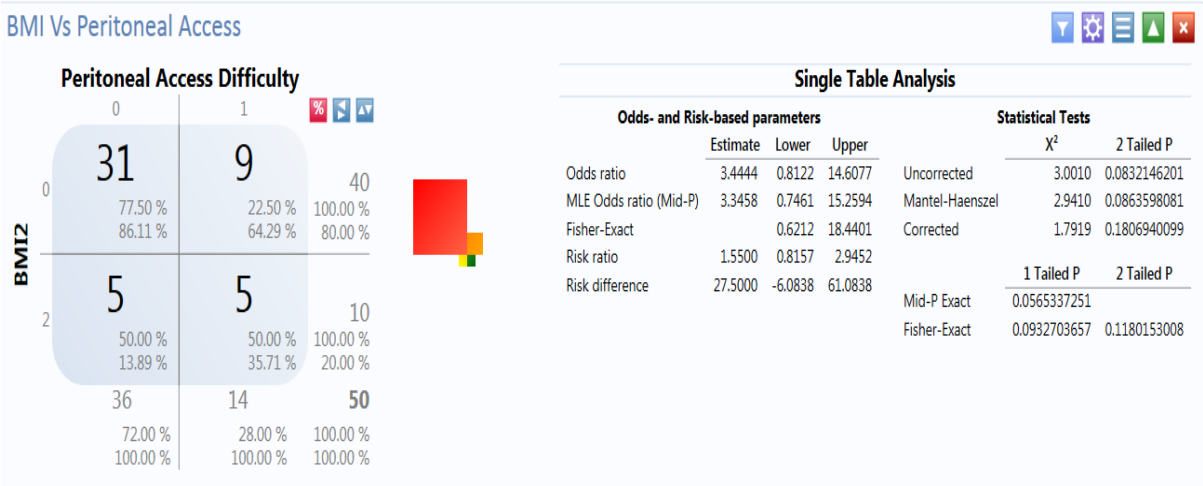


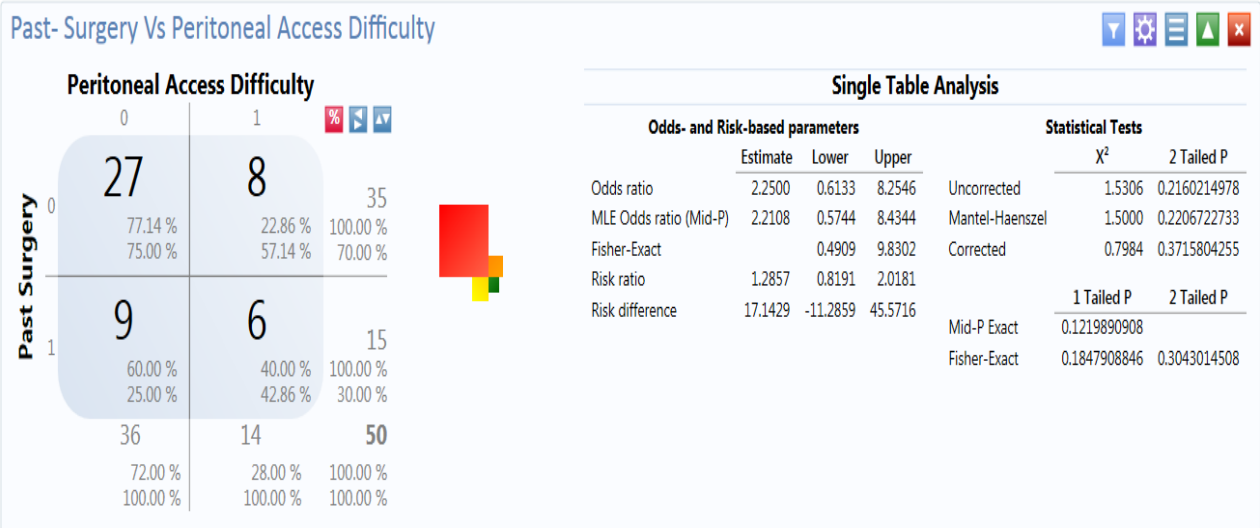
Table:8 BMI as Factor for difficult cholecystectomy

BMI Kg/m^2	Difficult Peritoneal access	Difficult Bed Dissection	Difficult GB Extraction	Duration of Surgery	Difficult Surgery
<30 (n=40)	9 (64%)	17 (73%)	9 (64%)	17 (73 %)	15 (37%)
>30 (n=10)	5 (36%) (p=0.05)	5 (26%) (p=0.3)	5 (36%) (p=0.03)	6 (26%) (p=0.17)	5 (50%) (p=0.24)

From the above results, it is evident that surgeons faced difficulty in accessing the peritoneal cavity and extraction of Gall bladder in persons with BMI > 30 kg/m^2.

PAST INTRA- ABDOMINAL SURGERY:

In this study, 15 patients had a previous history of Intra- abdominal surgery of which 6 (42.8%) had difficulty in accessing the peritoneal cavity. All patients had previous lower intra- abdominal surgery – Appendicectomy (4), Hysterectomy (4), LSCS (7). None of the patients had upper abdominal surgery



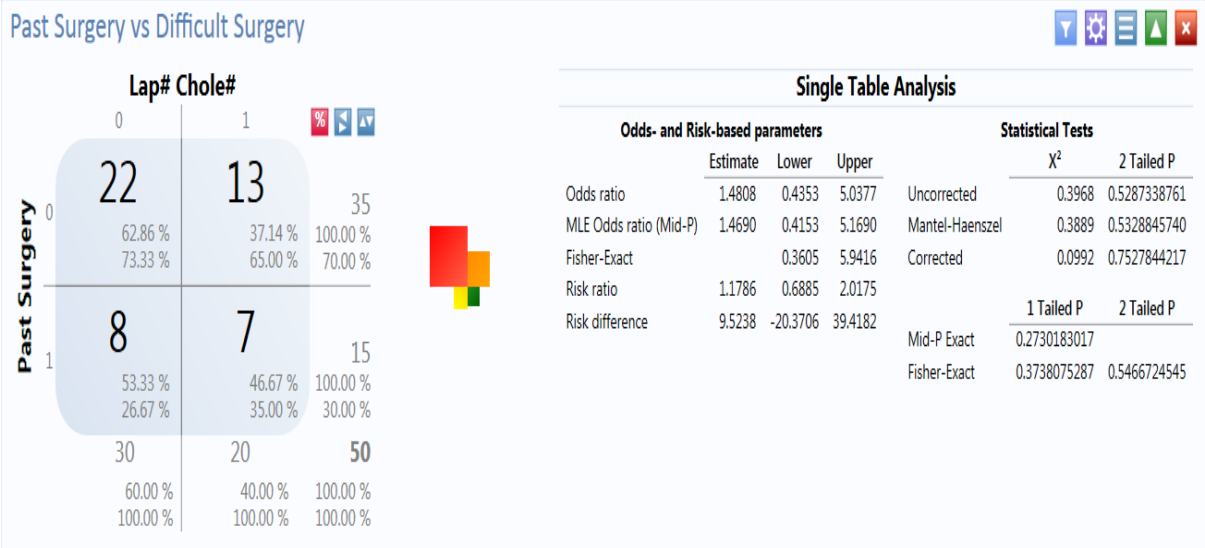


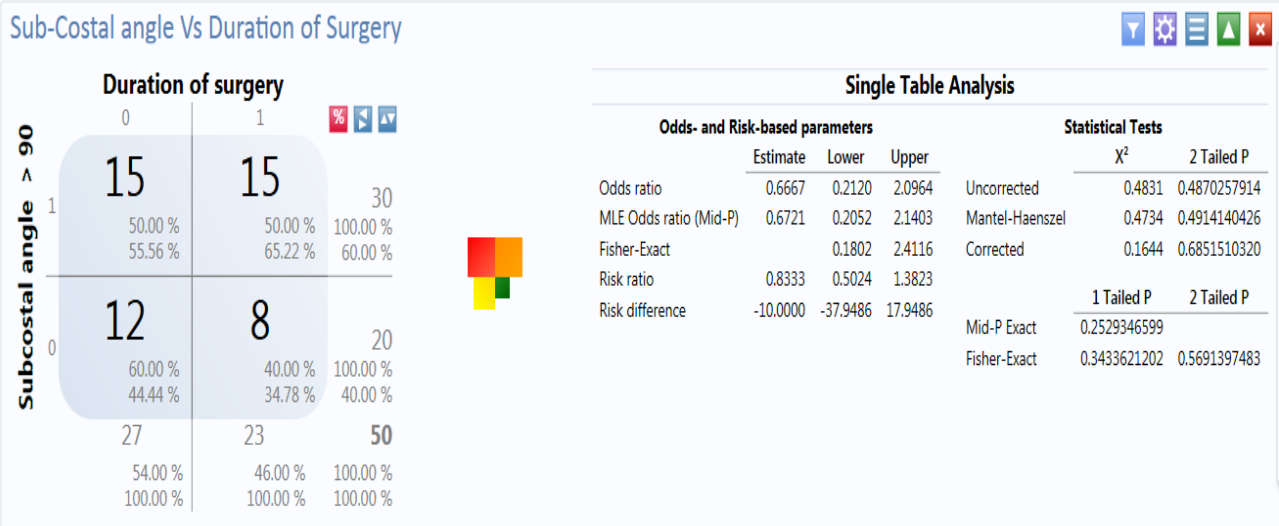
Table: 9 Past History of Surgery as Factor for difficult cholecystectomy

Previous Surgery	Diff. Peritoneal Access	Difficult Surgery
Yes (n=15)	6 (42%) p=0.12	7 (35%) p= 0.27
No (n=36)	8 (58%)	13 (65%)

SUB-COSTAL ANGLE:

20 subjects has sub-costal angle less than $90^{o,}$ 34.7% (8) of which had duration of surgery greater than 90 minutes.

Chi-Square tests:



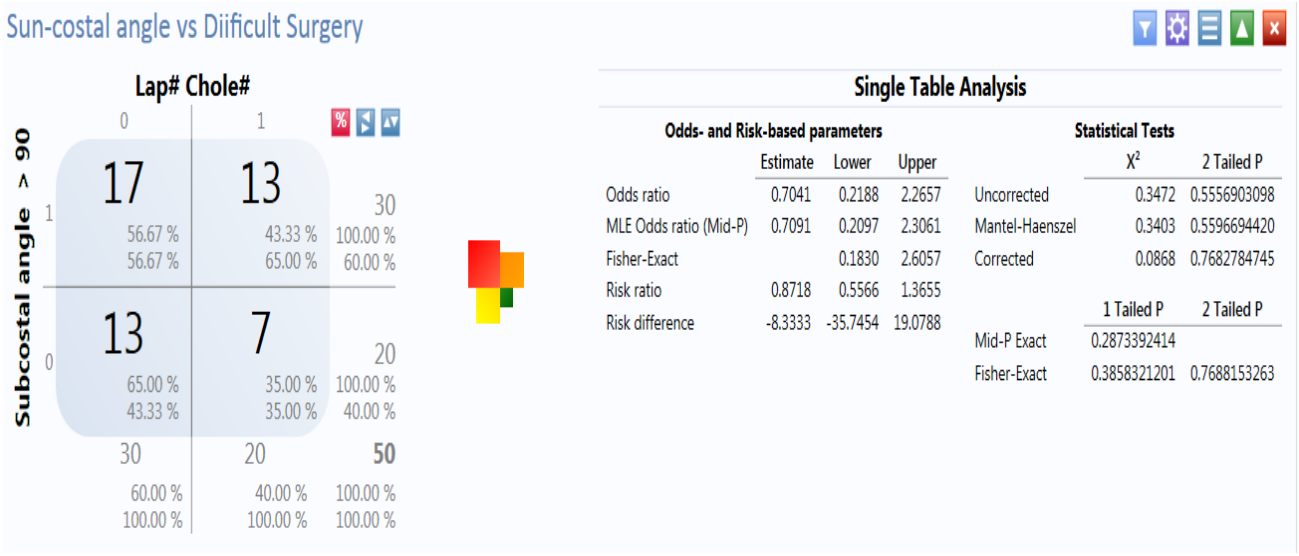


Table: 10 Sub-costal angles as Factor for difficult cholecystectomy

Sub-Costal Angle	Duration of Surgery	Difficult Surgery
>90^{o} (n=30)	15 (65%)	13 (65%)
<90^{o} (n=20)	8(35%) (p=0.25)	7 (35%) (p=0.28)

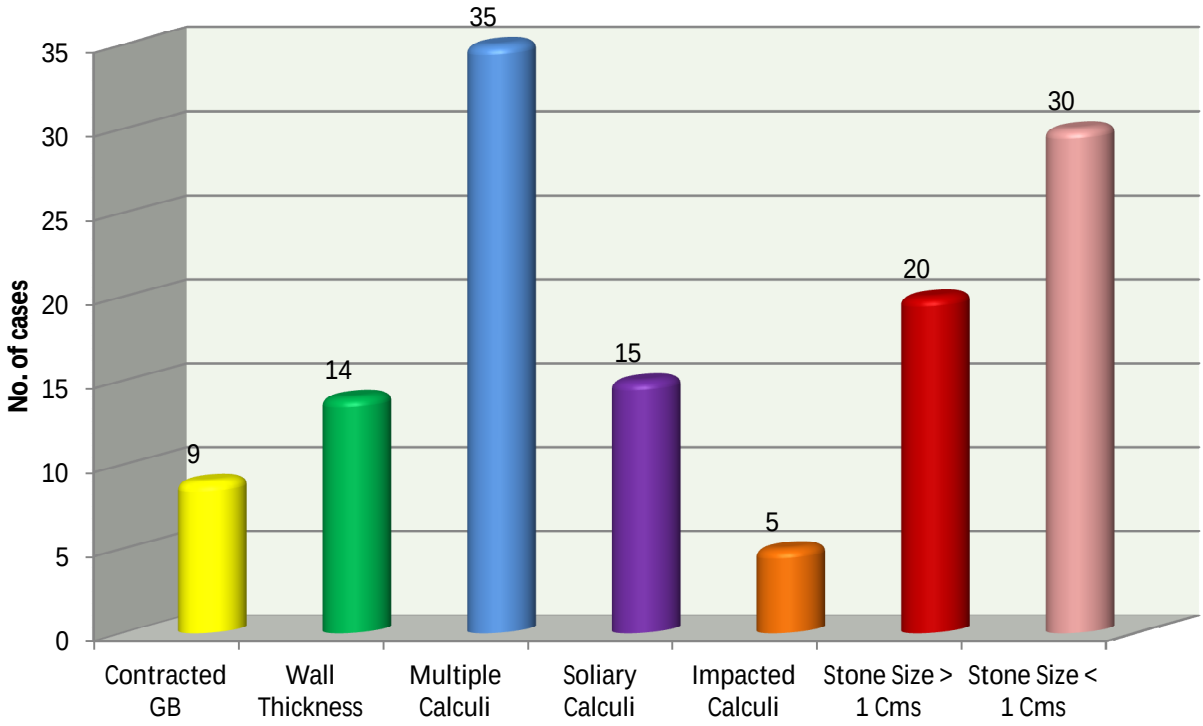
ULTRA-SONOGRAM PARAMETERS:

The Abdominal ultrasonogram findings of the 50 subjects are tabulated below.

Table: 11 Ultrasonological Parameters as a factor for difficult surgery

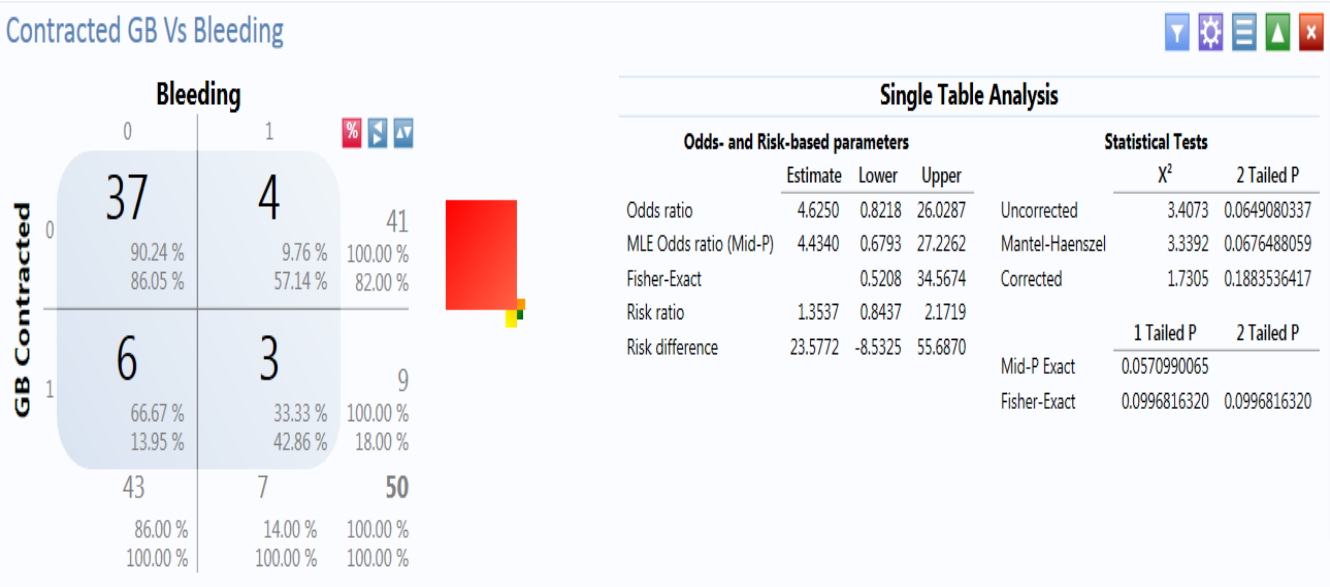
USG Parameters	**No. Of Patients**
Contracted GB	9
Wall Thickness	14
Multiple Calculi	35
Soliary Calculi	15
Impacted Calculi	5
Stone Size > 1 Cms	20
Stone Size < 1 Cms	30

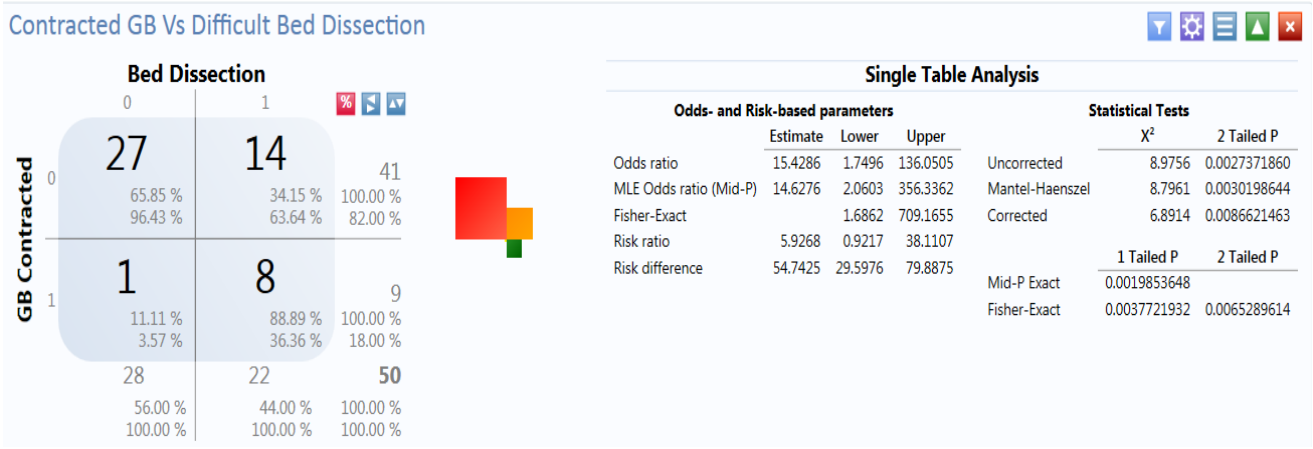
ULTRASONOLOGICAL FINDINGS



CONTRACTED GALL BLADDER:

9 of the 50 patients had contracted gall bladder.





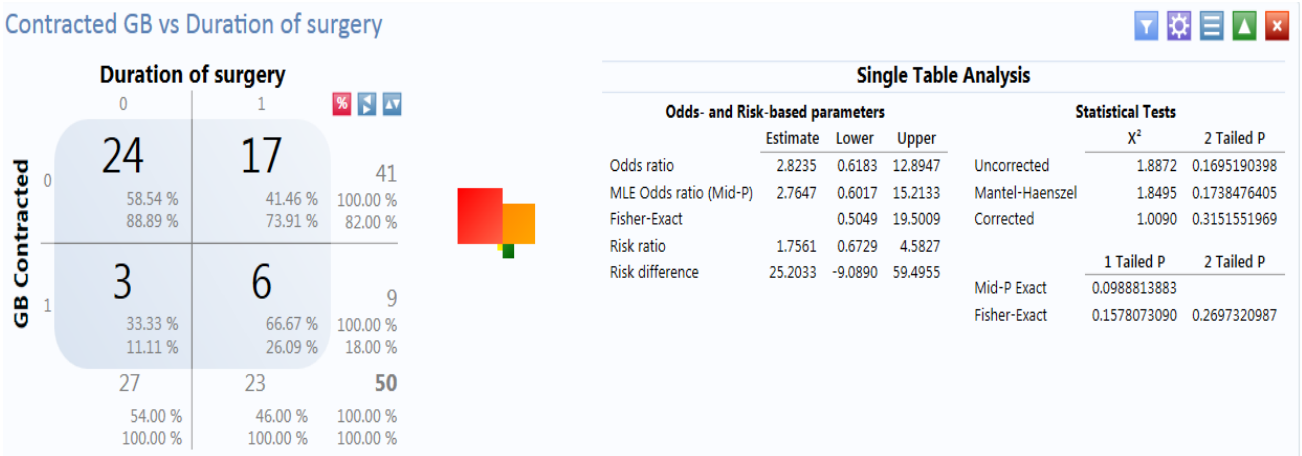


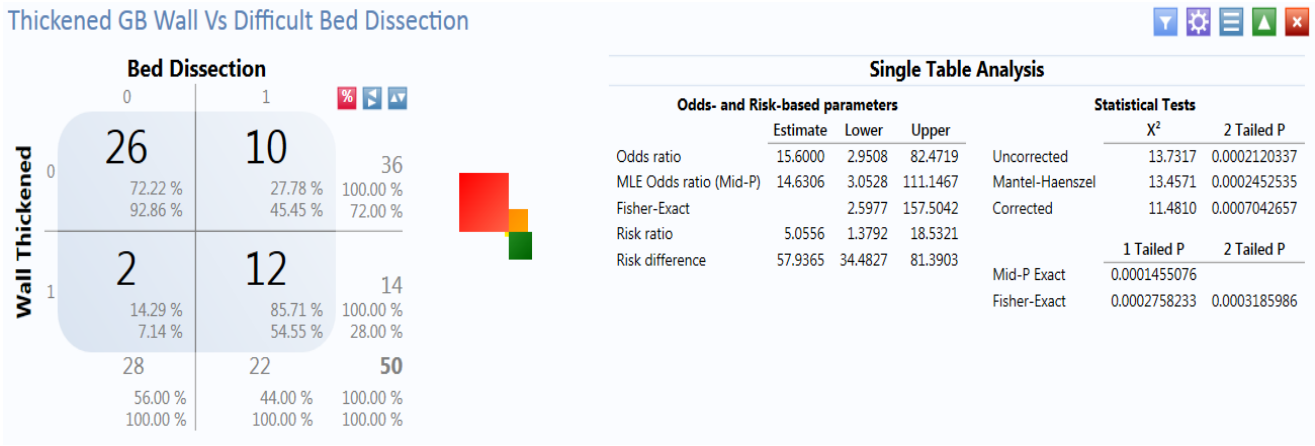
Table: 12 Contracted GB as a Factor for Difficult Laparoscopic Cholecystectomy

Contracted GB	**Bleeding**	**Difficult Bed Dissection**	**Difficult Extraction**	**Duration >90mins**	**Difficult Surgery**
Yes (9)	3 (33%) (p=0.05)	8 (89%) (p= 0.001)	7 (77%) (p=0.015)	6 (66%) (p=0.09)	7(78%) (p=0.008)
No (41)	4 (10%)	14 (34%)	13 (36%)	17 (41%)	13 (31%)

From above statistics, it was evident that the surgeons found contracted gall bladder to provide for difficult surgery by causing excessive bleeding and difficulty in bed dissection.

GALL BLADDER WALL THICKNESS:

Gall Bladder wall thickness was >4mm in 14 patients which indicated patient had chronic cholecystitis. This as a factor for difficult surgery is tabulated below.



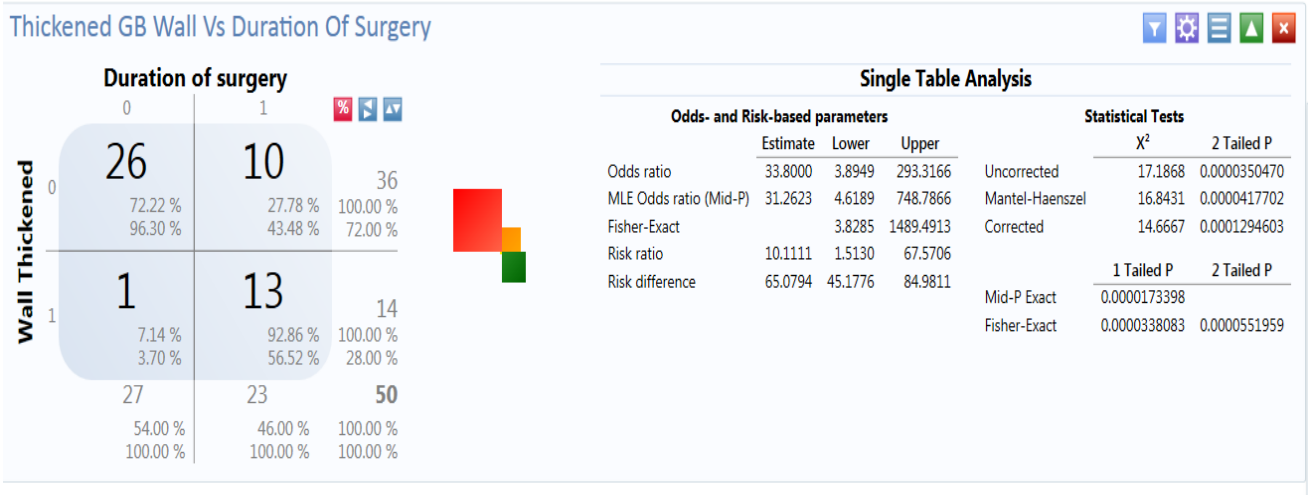


Table.13 Thickened GB wall as a Factor for Difficult Laparoscopic Cholecystectomy

Difficulty Faced	Bleeding	Difficult Bed Dissection	Difficult Extraction	Duration >90mins	Difficult Surgery
Thickeked GB Wall (n=14)	5 (71 %) p = 0.007	12(85%) p<0.0001	9 (69%) p=0.019	13 (92%) p<0.001	12 (85%) p<0.001
N Thickness <4mm (n=36)	2 (6%)	10 (27%)	13 (34%)	10 (28%)	8 (22%)

GALL STONES:

NO. OF CALCLUI:

Of the 50 patients, 35 had multiple Gall bladder calculi and 15 had solitary stone.

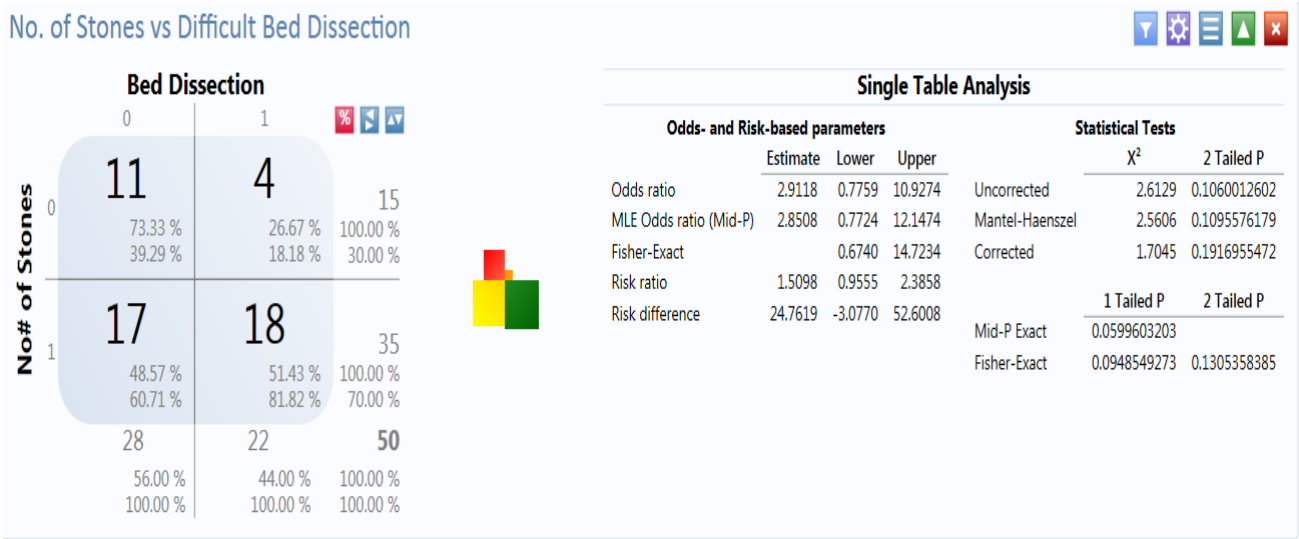


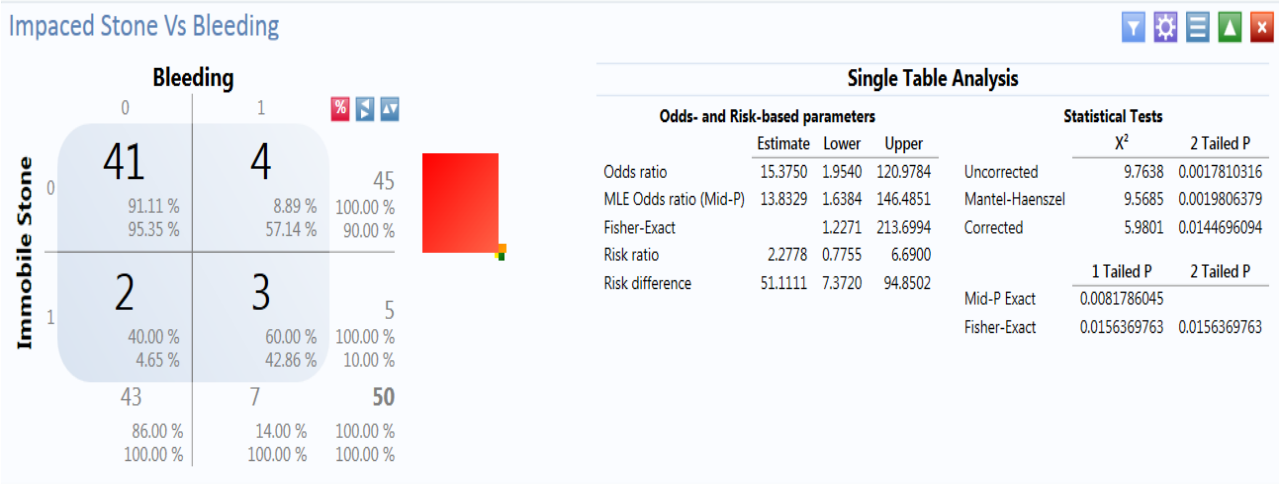
Table: 14 No. of stones as a Factor for Difficult Laparoscopic Cholecystectomy

Difficulty Faced	Bleeding	Difficult Bed Dissection	Difficult Extraction	Duration >90mins	Difficult Surgery
Multiple Calculi (35)	6 (17%) p=0.19	18 (52%) p=0.06	14 (42%) p=0.39	18 (51%) p=0.13	14 (40%) p=0.49
Solitary Calculi (15)	1 (7 %)	4 (26%)	7 (46%)	5 (33%)	6 (40%)

On the basis of above statistical analysis, multiple calculi proved to be problematic only during gall bladder bed dissection.

IMPACTED STONE:

Of the 50 patients, 5 patients had impacted stone while the rest had mobile stones determined by changing the patient position during ultrasonogram.



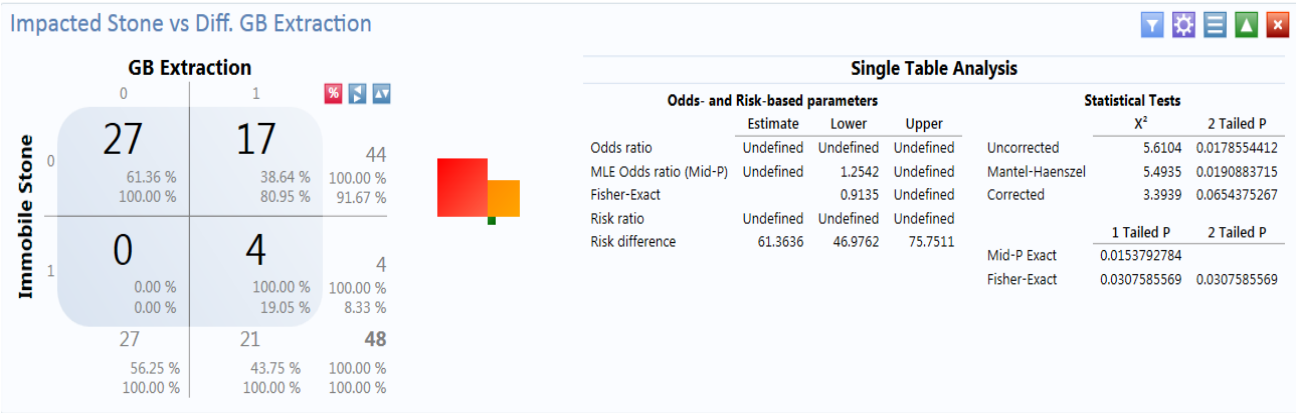


Table: 15 Impacted stone as a Factor for Difficult Laparoscopic Cholecystectomy

Difficulty Faced	Bleeding	Difficult Bed Dissection	Difficult Extraction	Duration >90mins	Difficult Surgery
Impacted stone (n=5)	3 (60%) p=0.008	3 (60%) p=0.24	4 (100%) p=0.015	4 (80%) p=0.072	14 (40%) p=0.49
Mobile Stone (n=45)	4 (9 %)	19 (42%)	17 (38%)	19 (42%)	6 (40%)

This analysis shows that there is correlation between impacted stone and moderate Bleeding during surgery and difficult extraction of gall bladder outside the abdomen.

SIZE OF THE CALCULI:

Of the 50 patients, 20 persons had Gall bladder stone size greater than 1cm which was considered to be an influencing factor for difficult surgery.

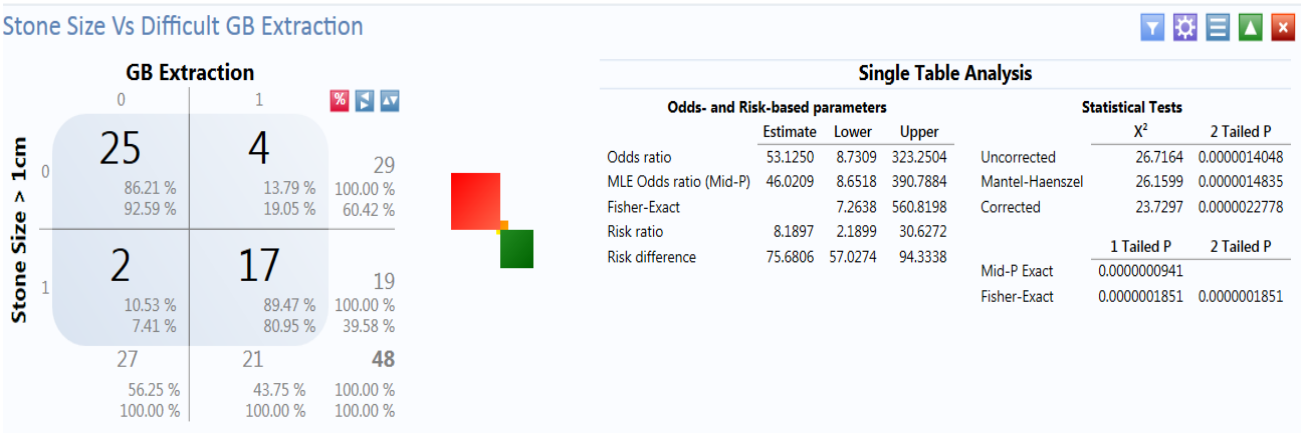
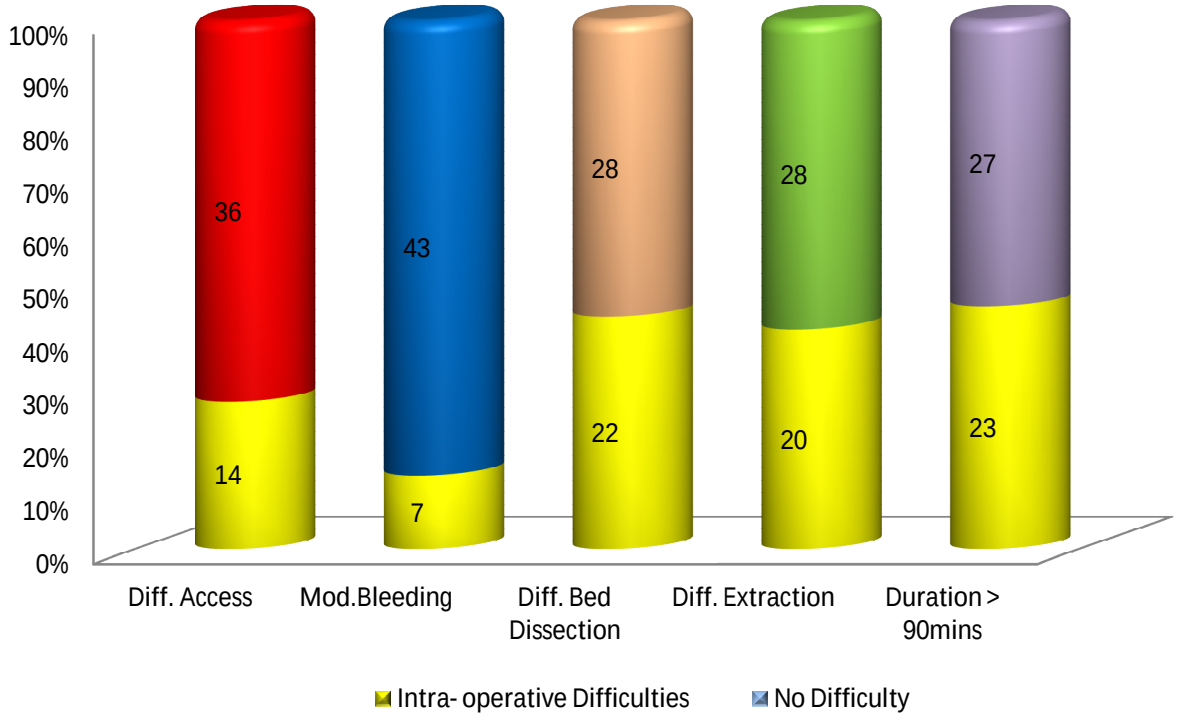


Table: 16 Stone size as a Factor for Difficult Laparoscopic Cholecystectomy

Difficulty Faced	**Bleeding**	**Difficult Bed Dissection**	**Difficult Extraction**	**Duration >90mins**	**Difficult Surgery**
Stone >1cm (n=20)	4 (20%) p=0.17	11 (55%) p=0.06	17 (90%) p<0.001	13 (66%) p=0.016	12 (60%) p=0.017
Stone < 1cm (n=30)	3 (10 %)	11 (26%)	4 (13%)	10 (33%)	8 (27%)

INTRA-OPERATIVE DIFFICULTIES:



PERITONEAL ACCESS:

Difficulty in accessing the peritoneal cavity like adhesions was encountered in 14 patients. One patient was converted to open cholecystectomy due to this reason.

Table: 17 Relationship Between Bleeding During Surgery and various parameters

Parameter	**No.**	**p Value**
BMI >30 (n=10)	5 (36%)	p=0.05
Past H/O Surgery (n=15)	6 (42 %)	p = 0.12

BLEEDING DURING SURGERY:

Of the 50 patients, moderate bleeding was encountered in 7 patients and none of the patients had severe bleeding.

Table: 18 Relationship Between Bleeding During Surgery and various parameters

Parameter	No.	p Value
Contracted GB (n=9)	3 (33%)	p=0.05
Thickened GB Wall (n=14)	5 (71 %)	p = 0.007
Multiple calculi (n=35)	6 (17%)	p=0.19
Impacted calculi (n=5)	3 (60%)	p=0.008
Stone size >1cm (n=20)	4 (20%)	p=0.17

GALL BLADDER BED DISSECTION:

Out of the 50 subjects, surgeons encountered difficult gall bladder bed dissection in 22 persons. LC in one female patient was converted to open cholecystectomy due to this difficulty.

Table: 19 Relationship between GB Bed dissection and various parameters

Parameter	No.	p Value
BMI >30 kg/m^2	5 (26%)	p=0.3
Contracted GB (n=9)	8 (89%)	p= 0.001
Thickened GB Wall (n=14)	12(85%)	p<0.0001
Multiple calculi (n=35)	18 (52%)	p=0.06
Impacted calculi (n=5)	3 (60%)	p=0.24
Stone size >1cm (n=20)	11 (55%)	p=0.06

GALL BLADDER EXTRACTION:

Of 48 patients who had successful LC, difficulty in extraction of the Gall Bladder out of the abdominal cavity was observed in 20 patients. 4 patients needed extension of the port incison for extraction while rest of the patients required removal of stones using forceps followed by extraction.

Table: 20 Relationship between GB Extraction and various parameters

Parameter	**No.**	**p Value**
BMI >30 kg/m^2	5 (36%)	p=0.03
Contracted GB (n=9)	8 (89%)	p= 0.001
Thickened GB Wall (n=14)	12(85%)	p<0.0001
Multiple calculi (n=35)	18 (52%)	p=0.06
Impacted calculi (n=5)	3 (60%)	p=0.24
Stone size >1cm (n=20)	11 (55%)	p=0.06

DURATION OF SURGERY:

Duration of surgery was prolonged (>90 mins) in 23 of the 50 patients who underwent LC.

Table: 21 Relationship between Duration of surgery and various parameters

Parameter	**No.**	**p Value**
BMI >30 kg/m^2	6 (26%)	p=0.17
Narrow Sub-costal angle	8 (35%)	(p=0.25)
Contracted GB (n=9)	6 (66%)	(p=0.09)
Thickened GB Wall (n=14)	13 (92%)	p<0.001

Multiple calculi (n=35)	18 (51%)	p=0.13
Impacted calculi (n=5)	4 (80%)	p=0.072
Stone size >1cm (n=20)	13 (65%)	p=0.016

CONVERSION TO OPEN CHOLECYSTECOMY:

Only 2 patients required conversion to open cholecystectomy.

- One was a Male patient with previous history of upper abdominal surgery where the surgeons had difficulty in accessing the peritoneal cavity due to dense adhesions.
- Second case was a 32 yr old obese female with thickened gall bladder wall and multiple calculi. It was converted to open due to difficulty in gall bladder bed dissection causing excessive bleeding.

DISCUSSION

Laparoscopic Cholecystectomy has become the gold standard treatment for symptomatic cholelithiasis with failure rates between 2 to 15 %. The conversion to open surgery does not strictly mean failure or a complication; it is seen as a measure to prevent further complication during the surgery. In this study of 50 patients undergoing LC, we have evaluated the factors, both clinical and Ultrasonological, which can be used to predict the difficulty in LC pre-operatively so that it can result in accurate planning of surgery and also proper counselling of the patient.

Analysing the age of the patients, most of them were equally distributed within the age range of 30 to 50 years whereas in Herman's series and Hanif series the majority of them were in the age group of 51- 60 yrs and 41-50 yrs respectively.[45] Categorising the age into two groups one less than 50 years and the other more than 50 years did not yield any significant correlation with the difficulties in surgery (p value = .45), which is similar to multiple studies in our review of literature. This is in contrast with the study by Eldar et al 46 which found age > 65 years, a significant independent factor associated with conversion. Schaefer et al [52] also identified age as a significant independent predictor of conversion. The observed disparity may be due to younger age of patients in the present study. The mean age of patients in the present study was

37.74 years. In Schafer's series mean age was 61.4 years with age range of 23-95 years [52].

The sex ratio of 1:2.4 was comparable to studies by Jagdish et al and Hanif et al. Male sex significantly predicted the conversion of laparoscopic cholecystectomy and was also found to be associated with significantly higher intraoperative severity grades (pvalue=0.04)

Eldar et al and Schafer et al [52] also found male sex to be a significant predictor of severity. The reason for higher rate of difficulty faced during LC in males can be explained from the observations that males have more intense inflammation and fibrosis resulting in difficult dissection of gall bladder bed. In our study too, the 50 % of the male patients had difficulty in gall bladder bed dissection.

Obese patients (BMI > 30 kg/m^2) had a significant effect on difficult peritoneal access (p=0.05) and gall bladder extraction (p=0.03) thus contributing to difficult chlecystectomy. This is compararble to observation by Philips et al and Schirmer et al.

History of previous intra- abdominal surgery did not have significant correlation with difficulties faced during LC especially getting peritoneal access (p=0.27) which is in contrary to the observations by Alpana et al and Darodhek et al. This can be explained on the basis that most of the patients had undergone lower abdominal surgery with only one having undergone upper abdominal

surgery. However, the one patient who had undergone upper abdominal surgery (Epigastric hernia) had to be converted to open due to dense adhesion.

Narrow sub-costal angle did not prove to be a significant predictive factor for difficult surgery (p=0.28) as observed in the study by Supe et al.

Ultra-sonological parameters had significant correlation with prediction of difficult cholecystectomy with each having influenced specific part of a surgery. In our study, Contracted gall bladder (n=9) had significant correlation with gall bladder bed dissection (p= 0.001) and bleeding during surgery (p=0.05).Thickened gall bladder wall (n= 14) proved to be a significant predictor of difficult surgery by having a good correlation with moderate bleeding during surgery ($p < 0.01$), gall bladder bed dissection ($p<0.001$) and which subsequently prolonged the surgery more than 90 mins ($p<0.001$).This can be explained by the fact that thick walled gall bladder and contracted gall bladder occurs most commonly in chronic cholecystitis which would have produced inflammation and fibrosis. Thickened GB wall was found to be most important predictor of difficulty in studies by Supe et al and Fried et al observations of which are comparable to our study.

Multiple calculi had a moderate correlation with difficult bed dissection (p=0.06). Impacted stone (n=5) also had a moderate correlation with bleeding during surgery ($p<0.008$) reason being fibrosis and inflammation in gall bladder due to impaction. Stone size greater than 1 cm (n= 20) was significantly associated with difficulty in extraction of gall bladder ($p<0.001$)

Only 2 patients had their LC converted to open surgery, one due to dense adhesion due to previous abdominal surgery, while the other was difficulty in gall bladder bed dissection. Our study had a conversion rate of 4 % which is comparable to other data available.[34, 44.] Reasons for conversion also correlated with observations made in study by Fried e*t al*[11.]

In our study, Thickened Gall bladder wall, contracted gall bladder, Stone size >1 cm significantly predicted the difficulty in Laproscopic cholecystectomy. Other factors which also played role were BMI >30 kg/m^2 and male gender. Fried et al's prospective study of 1,676 patients has similar observations except that our study had two extra parameters that were significant namely contracted gall bladder and stone size >1cm.

CONCLUSION

From our study we can conclude that various pre-operative predictors of difficult LC are present which influence various stages of the surgery which cumulatively or as a single factor make the surgery difficult for even the experienced laparoscopic surgeons.

The parameters that significantly correlate with the difficult surgery were Thickened gall bladder wall, contracted gall bladder , stone size >1 cm and to some extent BMI and Male gender.

Ultrasonological parameters play an upper hand in predicting the course of the surgery than by the clinical parameters. Hence a detailed Abdominal USG to look for these parameters would surely help in predicting the difficult surgery beforehand.

To conclude, prediction of difficult LC or conversion to open surgery will be helpful to both the patients and surgeons. For the patients, pre-op mental preparation can drastically reduce the post-operative stress and morbidity.

From surgeon’s point of view, patients with high risk for difficult LC could be operated by a experienced surgeon. Surgeons in the early phase of their training can mentally prepare for a difficult surgery there by negating intra-operative panic or can performs the LC under supervision of experienced surgeon.

On knowing the chances of difficult surgery or possibility of conversion to open prior to LC itself can enable the surgeon to convert to open cholecystectomy early if faced by any difficulties which can help in reducing the duration of surgery and subsequently the post-operative morbidity.

LIMITATIONS

The sample size of 50 patients in our study was relatively a smaller one which would have led on to sampling bias.

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ANNEXURE

INFORMED CONSENT FORM

Yourself Mr/Mrs/Ms…….…… are being asked to be a participant in the research study titled "**Clinical and ultrasonographical parameters as predictors of difficult laparoscopic cholecystectomy**" in CMC Hospital, Coimbatore, conducted by Dr.Thiruvalar Prabu Anand, Post Graduate Student in the Department of General Surgery, Coimbatore Medical College. You satisfy eligibility as per the inclusion criteria. You can ask any question you may have before agreeing to participate.

Research Being Done

Clinical and ultrasonographical parameters as predictors of difficult laparoscopic cholecystectomy

Purpose of Research

The aim of this study is to evaluate the role of preoperative clinical and ultrasound (US) scan findings as predictors of potential intra-operative difficulties, pitfalls, and complications during Laparoscopic cholecystectomy..

Procedures involved

In all selected patients, detailed history will be taken, physical examination will be done and particulars regarding other co morbid illnesses will be taken. Abdominal ultrasonography scan is performed within 24 hours before the surgery. Assess the difficulties faced during gall bladder surgery.

Decline from Participation

You have the option to decline from participation in the study existing protocol for your condition.

Privacy and Confidentiality

Privacy of individuals will be respected and any information about you or provided by you during the study will be kept strictly confidential.

Authorization to publish Results

Results of the study may be published for scientific purposes and/or presented to scientific groups; however you will not be identified.

Statement of Consent

I volunteer and consent to participate in this study. I have read the consent or it has been read to me. The study has been fully explained to me, and I may ask questions at any time.

---------- ----------

Signature /Left thumb impression Date

(volunteer)

---------- ----------

Signature of witness Date

STUDY DATA OF SUBJECTS

Name:…….. Age:……. Sex:…… I.P. No.:……

Study No:…….. Address:…..……
……
……
……

Height:……...Weight:……..…..BMI:……

Clinical History:

H/O Previous Abdominal Surgeries: Yes/No

Examination:

SubCostal Angle:

USG Findings:

S.No.	Criteria	Findings	Risk
1.	Gall Bladder		
2.	Wall Thickness		
3.	No. Of Stones		
4.	Stone Mobility		
5.	Stone Size		

INTRA-OPERATIVE:

Duration of surgery (in minutes): ……

Bleeding during surgery: Mild/ Moderate/Severe

Access to peritoneal cavity: Easy/ Difficult

GB bed dissection: Easy/Difficult

GB extraction: Easy/Difficult

Conversion to OC: Yes/No

Assessment of the Surgeon on the Difficulty of LC: Easy / Difficult

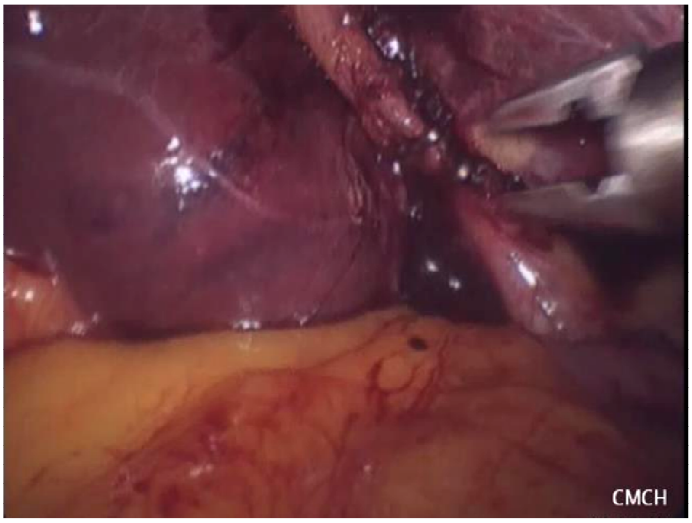
Objective assessment of the Difficulty of LC : Easy / Difficulty

LAPAROSCOPIC CHOLECYSTECTOMY

IN COIMBATORE MEDICAL COLLEGE HOSPITAL

EXPOSURE OF PORTAHEPATIS

IDENTIFICATION OF THE CYSTIC DUCT AND ARTERY



CLAMPING OF THE CYSTIC DUCT AND ARTERY

DIFFICULT GB BED DISSECTION WITH BLEEDING

S.No.	Name	Age	Sex	D.O.S.	Hospital No.	RHC Pain	Dyspepsia	Jaundice	BMI	Past Surgery	Sub costal angle > 90	GB Contracted	Wall Thickened	No. of Stones	Immobile Stone	Stone Size > 1cm	Duration > 90 min	Peritoneal Access	Bleeding	Bed Dissection	GB Extraction	Conversion to OC	Lap Chole Difficulty
1	Valliyammal	49	F	28-10-12	58560	y	n	n	0	0	1	0	0	1	0	0	0	0	0	0	0	0	0
2	Sundaram	48	M	06-11-12	66675	y	y	n	0	1	0	0	0	1	0	0	1	1	0	1	0	0	0
3	Hema	39	F	27-12-12	76602	y	n	n	0	0	1	0	0	0	0	1	1	0	0	0	1	0	1
4	Manju	46	F	12-01-13	1514	y	n	n	0	1	0	0	0	1	0	0	0	0	0	0	1	0	0
5	Raja	30	M	20-02-13	9847	y	n	n	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
6	Vijayalaksmi	26	F	11-04-13	17906	n	y	n	1	0	0	0	0	0	0	0	0	1	0	0	0	0	0
7	Lakshmi	36	F	23-02-13	2163	y	n	n	0	0	1	0	0	1	0	1	1	0	0	1	1	0	0
8	Sundari	62	F	28-02-13	7486	n	y	n	0	1	1	0	1	1	0	0	1	0	0	1	0	0	0
9	Amutha	42	F	07-03-13	11479	y	y	n	0	0	1	0	0	1	0	1	0	0	0	0	1	0	0
10	Vennila	35	F	12-03-13	13762	n	y	n	0	1	0	0	1	0	0	0	1	0	1	1	0	0	1
11	Periyanayaki	31	F	13-03-13	14118	n	y	n	0	0	1	0	0	1	0	0	0	0	0	0	1	0	0
12	Raman	52	M	21-03-13	15950	y	y	n	1	0	1	0	1	1	1	1	1	1	1	1	0	1	1
13	Ramani	52	M	21-03-13	15950	y	n	n	0	0	0	0	0	1	0	0	0	0	0	1	0	0	1
14	Shenbagam	25	F	10-04-13	20410	n	y	n	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
15	Suseela	27	F	10-04-13	19613	n	y	n	0	1	1	0	0	1	0	0	0	1	0	0	0	0	0
16	Asina	50	F	14-04-13	20852	y	y	n	0	0	0	1	0	1	0	1	0	0	0	1	1	0	0
17	Saraswathy	30	F	20-04-13	22680	y	n	n	1	0	1	0	0	0	0	1	1	1	0	0	1	0	1
18	Nallu	70	M	30-04-13	23803	y	y	n	0	1	0	1	1	1	0	0	1	0	1	1	0	0	1
19	Seetha	25	F	16-05-13	28224	n	y	n	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0
20	Lakshmi	42	F	18-05-13	28211	y	n	n	0	0	0	0	1	1	0	1	1	0	0	1	1	0	1
21	Chandralekha	31	F	25-05-13	29985	y	y	n	0	0	1	1	0	1	0	0	1	0	0	1	0	0	1
22	Valliyammal	75	F	30-05-13	23958	y	y	n	0	0	0	0	1	0	0	1	0	0	0	0	0	0	0
23	Radha	40	F	08-06-13	32856	y	y	n	1	0	1	1	0	0	1	1	0	1	0	0	1	0	0
24	Asina	27	F	13-06-13	34767	y	y	n	0	1	1	0	0	1	0	0	0	0	0	0	0	0	0
25	Jaise	21	F	20-06-13	30264	y	n	n	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0

26	Darwin Peter	15	M	28-06-13	37506	y	n	n	0	0	0	0	0	1	0	0	1	0	0	0	0	0	0
27	Valli	26	F	05-07-13	36515	y	n	n	0	0	0	0	0	0	0	1	0	0	0	0	1	0	0
28	Rangan	48	M	16-07-13	41306	n	y	n	0	0	1	1	1	1	0	1	1	0	1	1	1	0	1
29	Jamila	25	F	19-07-13	39643	n	y	n	0	0	1	0	0	1	0	0	0	0	0	0	0	0	0
30	Rajammal	50	F	20-07-13	38072	y	y	n	0	1	1	0	0	0	0	0	1	1	0	1	0	0	1
31	Mallika	48	F	22-07-13	33543	n	y	n	1	1	1	0	0	0	0	1	0	0	0	0	1	0	0
32	Shakila	31	F	23-07-13	42973	n	y	n	0	1	0	1	1	1	1	1	1	1	0	1	1	0	1
33	SakilaBanu	46	F	25-07-13	35642	n	n	n	0	0	1	0	1	0	0	1	1	0	0	1	1	0	1
34	Puspa	54	F	27-07-13	42932	y	n	n	1	1	1	0	0	1	0	0	1	0	0	1	0	0	0
35	Saravana Kumar	35	M	01-08-13	43987	n	y	n	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0
36	Muniammal	55	F	18-08-13	45966	y	y	n	0	0	1	0	0	1	0	0	0	1	0	0	0	0	0
37	Sathayveni	45	F	20-08-13	49896	y	n	n	0	0	1	0	1	1	0	1	1	0	0	1	1	0	1
38	Kanimozhi	34	F	21-08-13	48323	y	n	n	0	0	1	0	0	1	0	0	0	0	0	0	0	0	0
39	Saratha	55	F	22-08-13	50163	n	y	n	1	1	1	1	0	1	1	1	1	0	1	1	1	0	1
40	Johnson	52	M	31-08-13	52316	y	y	n	1	0	0	1	1	1	0	0	1	0	0	1	1	0	1
41	Nageshwari	40	F	31-08-13	50413	Y	n	n	0	0	1	0	0	1	0	1	0	0	0	0	0	0	0
42	Sabeetha	35	F	11-09-13	54596	n	y	n	0	0	0	0	0	1	0	0	0	1	0	0	0	0	0
43	Shanmugam	35	M	19-09-13	55357	n	y	n	0	1	1	1	0	0	0	0	0	0	0	1	1	0	1
44	Gobinath	26	M	31-10-13	65477	y	n	n	0	0	1	0	1	1	1	1	1	1	1	0	1	0	1
45	Nanjammal	52	F	07-11-12	64361	y	n	n	0	1	1	0	0	1	0	0	0	1	0	0	0	0	0
46	Stephan	55	M	18-12-12	76354	y	n	n	0	0	0	0	1	1	0	1	1	0	0	1	1	0	1
47	Kumarasamy	45	M	21-11-13	70072	n	y	n	1	0	0	0	0	1	0	0	0	1	0	0	0	0	0
48	Lakshmi	36	F	23-01-13	2163	y	y	n	0	0	1	0	0	1	0	0	0	0	0	0	0	0	0
49	Mary	50	F	05-09-12	49584	y	n	n	1	0	1	0	0	1	0	1	1	0	0	1	1	0	1
50	Vijayalaksmi	32	F	09-10-12	53160	y	n	n	0	1	1	0	1	1	0	0	1	1	1	1	0	1	1

1, y – YES 0, n – No RHC – Right Hypochondrium BMI – Body Mass Index